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MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—10TH YEAR. SYDNEY: SATURDAY, AUGUST 25, 1923. No. 8.

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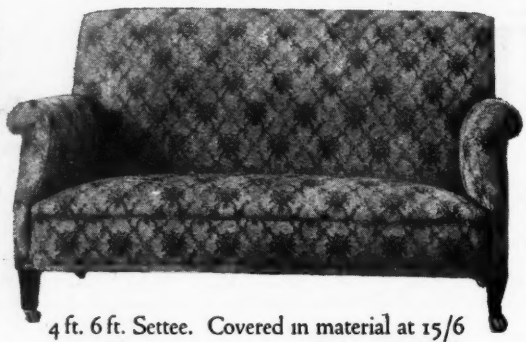

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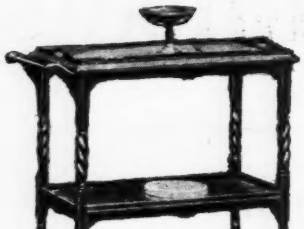

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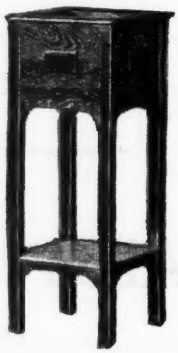

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A MODIFIED METHOD FOR THE MANUFACTURE OF "INSULIN."[1]

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AND

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AN essential factor in the ultimate success of "Insulin" therapy in diabetes will be the inexpensiveness of the product, for, since the administration must, so far as we can see at present, be continued throughout life, a price which in the case of other remedies would be of trivial importance, in this instance would be prohibitive and would result in the restriction of employment of "Insulin" to those patients who are in immediate and acute danger. Any discovery which lessens the expense of manufacture and enhances the yield from a given weight of pancreas, is therefore of practical importance.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 26, 1923.

The method of preparing "Insulin" which was originally devised by Collip was briefly as follows[2]:

To small volumes of 95% ethyl alcohol freshly minced pancreas is added in equal amount. The mixture is allowed to stand for a few hours with occasional shaking. It is then strained through cheese-cloth and the liquid portion at once filtered. The filtrate is treated with two volumes of 95% alcohol. This coagulates the greater part of the protein, leaving the "Insulin" in solution.

After allowing some hours for the coagulation of protein to be effected, the mixture is filtered and the alcohol removed by distillation *in vacuo*. Lipoids are removed from the aqueous residue by extraction with ether. The watery layer is further evaporated *in vacuo* until it is of pasty consistency. This residue is taken up in 80% alcohol and centrifuged to remove salts and coagulated protein. The clear supernatant fluid is delivered into several volumes of absolute alcohol which precipitates "Insulin" in an impure form.

It was subsequently ascertained by Doisy, Somogyi and Shaffer[3] that a great deal of loss of "In-

sulin" occurs during the initial extraction of the pancreas tissue with an equal volume of 95% alcohol and that the yield could be greatly enhanced by acidifying this mixture by the addition of thirty to forty cubic centimetres of concentrated sulphuric or hydrochloric acid per litre of minced pancreas, presumably because the immediate inactivation of pancreatic enzymes at this acidity protects the "Insulin" from digestion. These authors contributed the further important discovery that "Insulin" may be precipitated from aqueous solution in a comparatively pure condition by half-saturation with ammonium sulphate. These features of Doisy, Somogyi and Shaffer's method have been incorporated into the original method by the workers at the Toronto laboratories.[1]

The extraction of the tissue with an equal volume of acidified alcohol yields, after removal of the tissue by squeezing through cheese-cloth, a fluid which contains approximately 50% of alcohol. To this Collip adds a further two volumes of alcohol to coagulate the proteins by raising the final concentration of alcohol to about 80%. All of this alcohol must subsequently be removed by distillation, compelling subsequent rectification of the distillate to recover the alcohol, a certain wastage of this expensive solvent and a considerable expenditure of time. It occurred to us that the same end, namely coagulation of proteins by increase of the alcoholic strength, might be achieved just as readily by removal of water from the original extract as by the addition of alcohol to it. This we can accomplish by taking advantage of the fact that anhydrous sodium sulphate combines with 1.27 times its own weight of water to form the crystaline sulphate, $Na_2SO_4.10H_2O$. We accordingly add to the extract containing 50% of alcohol a sufficient amount of anhydrous sodium sulphate or of the exsiccated sulphate, $Na_2SO_4.H_2O$, to remove four-fifths of the water, rendering the alcoholic concentration a little over 80%, as in Collip's method, but in a much smaller volume of fluid, so that much less time is consumed in distillation and the rectification of recovered distillates and much less alcohol is wasted. The sodium sulphate can be completely recovered by ignition and employed for the same purpose again. The further stages of our process closely resemble those which are now employed at Toronto. The full details of our method are as follow:

Six pounds (about 2.75 kilograms) of hard frozen pancreas tissue are minced and mixed at room temperature with an equal volume of 95% alcohol containing thirty cubic centimetres of concentrated hydrochloric acid (36%) per litre. Fifty cubic centimetres of ether are added to the mixture to facilitate cytolysis of the cells. The mixture is placed in a receiver of squat form and vigorously stirred by a mechanical stirrer for four hours. The mixture is then strained through cheese-cloth and to the strained, but not filtered extract 10% sodium hydrate is added until the mixture is faintly alkaline to litmus. To this mixture is then added one kilogram of anhydrous (or a proportional amount of exsiccated) sodium sulphate for every three litres of extract. After stirring for a brief period and ascertaining that the reaction is still faintly alkaline, this mixture is allowed to stand overnight at a low temperature. It is again stirred and then centrifuged in stoppered tubes. It forms three layers in the centrifuge tube, namely a lower layer of crystals of sodium sulphate, a middle layer of fragmented tissue and coagulated protein and an upper layer of clear solution. This upper layer and that portion of the middle layer which flows most readily, are decanted from the tube and immediately filtered. The filtrate is placed in a vaccum still and the alcohol distilled off at about 35° until frothing interferes with the distillation, when the residual alcohol may be removed by blowing warm air over the mixture in shallow pans, in a Hearson serum-drier or similar apparatus. The volume having been reduced to about one-tenth of the original volume of pancreas hash (300 cubic centimetres), the fluid is extracted twice with ether to remove the fat. The excess of ether in the aqueous layer is removed by blowing warm air over the fluid and the volume is further reduced to about 150 cubic centimetres. Sufficient alcohol is now added to render the mixture 76% to 80% by volume. Salts are precipitated and some protein. The mixture is centrifuged and the fluid decanted from the subnatant crystals and saturated salt solution. The pH of this fluid is adjusted to between 5 and 6, methyl orange or B.D.H. universal indicator being used and an aliquot sample being titrated. Five volumes of absolute alcohol or of a mixture of four parts of absolute alcohol to one of ether are now added and the mixture in tall glass cylinders is set aside in a cool place until the flocculant white precipitate has settled. The greater part of the fluid can usually be poured off without loss of the precipitate. The remainder with the suspended precipitate is centrifuged in stoppered tubes and the ether-alcohol mixture is poured off. The precipitate still remaining in the tubes, is drained as free as possible of alcohol and, without permitting the precipitate to dry (which appears to hinder its solution) somewhat less than the calculated volume of sterile saline solution is added and poured from tube to tube until all the "Insuin" is dissolved in it. The small percentage of adherent alcohol augments the solubility of the product. The solution is poured into a sterile graduated cylinder, the pH adjusted to about 7.0 with sterile alkali and the volume made up to the calculated magnitude by the addition of sterile saline solution. Either of two methods may now be adopted. The one is to add one cubic centimetre of ether to every fifty cubic centimetres of fluid, to shake the mixture thoroughly in a stoppered cylinder and to introduce from a sterile hypodermic syringe into sterile ampoules or, alternatively, the fluid without previous addition of ether may be introduced into the ampoules and one drop of ether placed in each ampoule before sealing. The solution may be more effectively sterilized by filtration through a Berkefeld filter, but this involves some loss of potency. The product may be further purified, if desired, by precipitation from the aqueous solution by half-saturation with ammonium sulphate, followed by extraction of the precipitate

with from 70% to 80% alcohol and reprecipitation with alcohol-ether mixture.

The yield is from 50 to 60 two-kilogram "rabbit units" or 150 to 180 of the new units per pound of untrimmed pancreas tissue, corresponding to one two-kilogram "rabbit unit" from every eight grammes of untrimmed pancreas. This yield could doubtless be considerably enhanced by the use of filter-presses to remove the last portions of extract from the residue of pancreas tissue and also to remove the alcoholic fluid from the sodium sulphate crystals, instead of employing the centrifuge for this latter purpose.

We have found as indicated above that frozen pancreas may safely be employed in place of the fresh tissue, a fact which will considerably facilitate the collection and storage of the tissue in abattoirs. We have not detected any notable defect of yield even after storage for one week in the refrigerating room, but the temperature must be below freezing-point and the tissue should be spread in a thin layer upon oiled paper to insure a rapid fall of temperature after removal from the animal.

We wish to acknowledge our indebtedness to Miss M. Cheadle and Mr. H. R. Marston for assistance which they rendered in the early stages of our experiments and also our great indebtedness to Mr. F. W. Fillmore, Manager of the Metropolitan Abattoirs, and Dr. P. A. Burridge, Veterinarian at the Abattoirs, for their generous and enthusiastic collaboration, without which the successful prosecution of the investigations reported in this and the two following papers would have been impossible. Finally we wish to express our indebtedness to Professor J. J. R. Macleod and the committee in charge of the "Insulin" patents of the University of Toronto for the permission extended to us to manufacture "Insulin" and for the opportunity thus afforded us of participating in the investigation of the physiological and therapeutic effects of this most interesting substance.

Summary.

A modification of Collip's method of preparing "Insulin" is described, in which concentration of the alcohol in the original 50% alcoholic extract of the pancreas is achieved by removal of water from the extract instead of by addition of alcohol. This is accomplished by the addition of anhydrous sodium sulphate to the extract and subsequent separation of the fluid from the crystals. A considerable saving of time and reagents is thus effected and the yield of "Insulin" which is obtained, compares very favourably with that obtained by other, more expensive methods.

References.

(1) "New and Non-Official Remedies; Insulin," American Medical Association; *The Journal of the American Medical Association*, June 2, 1923, page 1617.

(2) Collip, J. B.: Proceedings of the American Society of Biological Chemists; *The Journal of Biological Chemistry*, 1923.

(3) Doisy, Somogyi and Shaffer: Proceedings of the American Society of Biological Chemists; *The Journal of Biological Chemistry*, 1923.

THE ABSENCE OF DIRECT EFFECT OF "INSULIN" UPON THE OXIDATION OF DIACETIC ACID IN MICE.[1]

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THE deficient oxidation of glucose in diabetics is accompanied by a proportionately deficient oxidation of diacetic acid which appears as an intermediate product in the oxidation of fats and is normally consumed, as rapidly as it is produced, to the final products of oxidation, namely CO_2 and H_2O. The accumulation of diacetic acid in the blood of the diabetic leads to the acidosis and ketonuria which are so characteristic of the more advanced stage of the disease.

A similar accumulation of diacetic acid may be induced in a normal individual by mere carbohydrate starvation, that is to say by subsisting upon a diet composed wholly of proteins and fats, especially if the latter form a large proportion of the diet. Hence it has come to be believed that in our tissues the oxidation of diacetic acid and that of glucose are so related that the diacetic acid can only be oxidized if glucose is simultaneously oxidized or, in other words, as Rosenfeldt has expressed it, that "the fats are burnt only in the fire of the carbohydrates." This opinion has obtained substantial support from the observation of Shaffer[6] that diacetic acid is not oxidized by alkaline hydrogen peroxide excepting in the presence of an equimolecular proportion of glucose, the special significance of this observation arising out of the fact that, as Dakin has shown, oxidations in the body usually pursue the same course as oxidations by alkaline peroxide *in vitro*.[5] Shaffer has furthermore shown[6] that, even in diabetics, acidosis and ketonuria do not develope unless the glucose which is actually oxidized, falls below the molecular equivalent of the total diacetic acid which may be derived from the constituents of the diet, chiefly the fats, but also in some proportion the proteins as well.

The discovery by Banting and Best and by Macleod and co-workers that "Insulin" rapidly diminishes the hyperglycæmia in diabetics and also raises the respiratory quotient, shows that "Insulin" does actually promote the consumption of glucose and, as we might anticipate from the above considerations, the acidosis and ketonuria are likewise and simultaneously diminished. The natural inference is that the oxidation of carbo-hydrates induced by "Insulin" incidentally promotes the oxidation of diacetic acid. This inference, however probable, has nevertheless as yet remained an inference and it appeared desirable to put it to the test of experiment and to inquire whether "Insulin" may not after all reduce acidosis by promoting the oxidation of diacetic acid directly, as well as indirectly through promotion of the oxidation of carbohydrates.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 26, 1923.

This inquiry appeared to be the more necessary because we have not infrequently noticed and Macleod has also observed[3] that when a dose of "Insulin" considerably in excess of the lethal dose is administered to an animal (rabbit) the effect may not be lethal and indeed, in some instances, only very mild symptoms of intoxication may supervene, while animals in receipt of smaller doses die or exhibit the convulsions typical of an overdosage of "Insulin." Now the toxic effect of an overdose of "Insulin" is related to the attainment of a certain degree of hypoglycæmia and hence it was at first supposed to be due to acidosis following upon such rapid and complete exhaustion of available carbohydrates that there is no longer a sufficiency of glucose available to accomplish the oxidation of diacetic acid which is still arising from the fats. At the present moment this interpretation of the toxicity of overdoses of "Insulin" must be held somewhat in doubt, for while certain of the symptoms of intoxication are reconcilable with and possibly arise from this origin, others point rather clearly towards cerebral excitation, a factor which was specially prominent in a case recently reported of overdosage in a human subject.[4] The peculiar immunity of occasional animals from the toxic effects of gross overdosage might nevertheless conceivably originate in a direct promotion of the oxidation of diacetic acid by these very large doses of "Insulin" compensating for the inadequacy of glucose to accomplish such oxidation indirectly.

In order to investigate this question it was necessary in the first place to deplete the carbo-hydrate reserves of the animals so thoroughly that there could be little or no opportunity for promotion of the oxidation of diacetic acid by concurrent oxidation of glucose. To achieve this end mere starvation is not sufficient, because the liver and muscles still retain carbo-hydrate reserves when actual death from starvation is imminent. Starvation assisted by administration of phloridzin, however, very rapidly accomplishes depletion of the carbo-hydrate reserves.

Preliminary experiments had shown us that the lethal dose of phloridzin followed by twenty-four hours of starvation in mice lies somewhat above twenty milligrammes for a mouse of twenty to twenty-three grammes weight. Doses of sixteen milligrammes of phloridzin[1] were administered to a series of mice which were thereafter starved for twenty-four hours. The lethal dosage of diacetic acid for these animals was then determined with and without concurrent administration of "Insulin."

The diacetic acid was prepared from pure ethyl aceto-acetate by the method of Creosole[5] and was kept at a low temperature in ether solution. When required known volumes of this solution were evaporated and the acid residue, consisting of aceto-acetic acid and a little unchanged ethyl aceto-acetate, was dissolved in physiological saline solution and administered to the animals hypodermically.

The lethal dose of this preparation of diacetic acid was determined by administering graded doses to

[1] We are very greatly indebted to Professor W. A. Osborne for supplying us with the phloridzin which we employed.

DIACETIC ACID ONLY.			DIACETIC ACID *PLUS* INSULIN.			
Mouse.	Dose of Impure Diacetic Acid. Milligrammes.	Results.	Mouse.	Dose of Impure Diacetic Acid. Milligrammes.	Dose of "Insulin." "Rabbit Units."	Results.
A.	200	Coma, followed by death during night.	B.	200	0.07	Coma, followed by death; "air hunger."
C.	200	Coma, death in ten minutes.	D.	200	0.07	Coma, followed by death.
E.	200	Coma, followed by death during night.	F.	130	0.07	Coma, recovery.
G.	150	Survived.	H.	150	0.1	Coma, death in a few hours.
I.	150	Recovered, but died 48 hours later, fed after 18 hours.	J.	Somewhat less than 150, a portion of dose having been lost.	0.1	Coma, followed by typical "Insulin" convulsions. Recovered.
K.	120	Died 24 hours later, fed after 18 hours.	L.	90	0.1	Coma, followed by typical "Insulin" convulsions. Recovered.

Sixteen milligrammes of phloridzin injected into each mouse followed by starvation for twenty-four hours.

a series of mice which had been previously phloridzinized and starved for twenty-four hours. It proved to be somewhat in excess of 150 milligrammes for a mouse of sixteen to twenty grammes weight. The lethal dose of "Insulin" was similarly determined and found to be a little more than one-tenth of the dose which is lethal for a two-kilogram rabbit. Lethal and sub-lethal doses of diacetic acid and sub-lethal doses of "Insulin" were then injected simultaneously into starved phloridzinized mice. A series of control animals of similar weight received like doses of diacetic acid but no "Insulin."

The results set out in the table on page 192 were obtained.

These results reveal no appreciable diminution of the toxicity of diacetic acid in consequence of the concurrent administration of "Insulin." In fact in the case of Mouse H, in which the dose of diacetic acid was on the border line between the lethal and sub-lethal dose, the administration of "Insulin" appeared rather to augment than diminish the effects of the diacetic acid, since two control mice, G and I, which received the same quantity of diacetic acid survived for forty-eight hours or more, while Mouse H died within a few hours after the administration of the diacetic acid together with "Insulin."

The "Insulin" which was used in these experiments, had been kept at a low temperature for three or four weeks. It may possibly not have retained the full potency determined by the original standardization with rabbits and we do not desire, therefore, to insist upon the accuracy of the ratio of the lethal dose of "Insulin" for phloridzinised mice to that for rabbits, which is indicated by the above estimates. The "Insulin," nevertheless, was definitely potent since two of the mice which received sub-lethal doses of diacetic acid, namely the mice J and L, together with a large dose of "Insulin" (one-tenth of the lethal dose for a two-kilogram rabbit) displayed the convulsions typical of overdosage with "Insulin." In the other animals the effects of the "Insulin" were less or were masked by the coma produced by the diacetic acid and no convulsions were observed. This fact shows that the convulsions due to overdosage of "Insulin" do not arise from acidosis due to accumulation of diacetic acid consequent upon excessive hypoglycæmia, for in that event they should occur all the more readily in the animals receiving the higher dosages of diacetic acid. On the contrary, while those animals which received lethal doses of diacetic acid, whether treated with "Insulin" or not, displayed typical "air-hunger" with slow, deep inspiratory gasps, none of them exhibited convulsions at all resembling those arising from overdosage of "Insulin."

Conclusions.

1. No appreciable extent of direct oxidation of aceto-acetic acid by "Insulin" could be demonstrated in phloridzinised animals.

2. The convulsions which accompany "Insulin" hypoglycæmia, are not due to acidosis caused by accumulation of aceto-acetic acid.

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(1) Creosole: *Berichte der deutschen chemischen Gesellschaft*, 1872, Band XV., *Seite* 1327.

(2) Dakin, H. D.: "Oxidations and Reductions in the Animal Body," London, Second Edition, 1922.

(3) McCormick, Macleod, Noble and O'Brien: *Journal of Physiology*, 1923, Volume lvii., page 234.

(4) "Some Clinical Results of the Use of 'Insulin,'" A Report to the Medical Research Council, *The British Medical Journal*, April 28, 1923, page 737.

(5) Shaffer, P. A.: *The Journal of Biological Chemistry*, 1921, Volume xlvii., pages 433 and 449; Volume xlix., page 143.

THE TREATMENT OF DIABETES BY "INSULIN."[1]

BY C. T. CHAMPION DE CRESPIGNY, D.S.O., M.D., M.R.C.P.,

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OWING to the kindness of Professor Brailsford Robertson who has supplied me with "Insulin" prepared in his Department in the University of Adelaide, I have had the opportunity of using this material in several selected cases of diabetes within the last few months.

The "Insulin" has been so standardized that one cubic centimetre is equivalent to one two kilogram "rabbit unit." It is issued in ampoules, each containing one cubic centimetre. The method of administration has been by injection beneath the skin of the arm, forearm or thigh except when for experimental purposes a few doses were given by the mouth.

Sometimes the injections are rather painful. Apart from this no ill effects have been met with.

"Insulin" should, if possible, be stored in a cool place, preferably on ice.

All the patients to whom "Insulin" has been given, have been under observation for some time before its administration and they have had their diet carefully controlled before and during its use. In these notes special stress will be laid upon the easily observed results of treatment, *id est* improvement of patients' well being, strength, weight, power to assimilate carbo-hydrates and diminution of acidosis.

With the biochemical changes caused by "Insulin," which are difficult to ascertain in actual practice, I shall not so much concern myself.

Case I.—A.C., *ætatis* thirty-five, is a patient of Dr. Dean Dawson and has been treated in consultation with Dr. Dawson. Having suffered from severe diabetes for three years, this man was very weak and emaciated. In spite of careful dieting his carbo-hydrate tolerance was almost nil. He showed a moderate degree of acidosis (his alveolar carbon dioxide was twenty-five millimetres of mercury). His weight on January 31, 1923, was 45.5 kilograms (100 lbs.) when he entered hospital for the purpose of treatment by "Insulin."

Table I., setting out the careful observations made by Dr. Ray Hone, shows the effect of treatment.

To this may be added the following brief observation of the effect of "Insulin."

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 26, 1923.

TABLE I.

Showing a study of the metabolism of this patient carried out by Dr. Ray Hone in Professor Robertson's Laboratory, with Professor Robertson's note.

Date.	Carbohydrate in diet in previous 24 hours. Grammes.	"Insulin." c.cm.	Alveolar CO_2 Tension.	Blood Sugar at Noon.	Urine Collected During Previous Twenty-four Hours.							
					Volume of Urine. c.cm.	Diacetic Acid.	Total Titratable Acidity. c.cm.	Total Ammonia. Milligrammes per 100 c.cm.	Total Nitrogen. Milligrammes per 100 c.cm.	Ammonia / Nitrogen × 100	Total Glucose. Grammes.	Dextrose / Nitrogen.
1923.												
Jan. 31 ..	25	None	25	0.24%	3000	+	696	1.14	20.3	5.6	81	
Feb. 1 ..	25	1 before breakfast, 1 twelve hours later	25	0.18%	3300	+	726	0.66	18.3	3.6	49.5	1.34
Feb. 2 ..	25	2 as above	25	0.25%	3700	None	740	1.85	18.2	1.2	9.3	
Feb. 3 ..	25	2 as above	25		3400	None	680	1.02	16.2	6.3	Trace	
Feb. 4 ..	25	2 as above			3000	None	510	1.50			Trace	
Feb. 5 ..	25	2 as above		0.17%	2800	None	896	9.84	18.1	4.6	Trace	
Feb 6—16..		2 per diem										
Feb. 17 ..		2 per diem		0.14% at 3 p.m.								
Feb. 18 ..		1 by mouth in a.m., 1 hypodermically in afternoon		0.22% at 3 p.m.								
Feb. 19 ..		2 hypodermically		0.18% at 3 p.m.								
Feb. 20 to Mar. 10 ..		2 hypodermically per diem as above										
Mar. 11 to 14		None										
Mar. 15 ..		None		0.19% at 3 p.m.								
Mar. 16 to 21		2 hypodermically per diem										
Mar. 22 ..		2 as above		0.15% at 3 p.m.								

The dextrose-nitrogen ratio on February 1 is the ratio of the excess of output over input of carbo-hydrate to the total output of nitrogen. Note the failure of oral administration on February 18 and the slight rise of blood sugar after five days without treatment ending March 15, followed by the considerable fall after reinauguration of treatment.

Treatment.	Date.	Carbo-hydrate. Grammes.	Protein. Grammes.	Fat. Grammes.	Calories.	Glucose in Urine. Grammes.	Blood Sugar.
Without "Insulin"	31/1/23	25	75	32	684	84	0.29%
With "Insulin" (1 "rabbit unit" at 7 a.m. and 7 p.m.)	19/2/23	40	79	75	1555	nil	0.14%

Since leaving hospital this patient has continued to improve. His weight has increased by seven kilograms and he feels stronger and better in every way. He has been doing some light work. However, his urine contains sugar from time to time with a daily ration of thirty grammes of carbo-hydrate.

Case II.—R.M., *ætatis* forty, was sent to me by Dr. Riddell, of Kapunda. This man, a pastoralist, had suffered from diabetes for two years when I treated him in January, 1923. He was then sugar-free on a daily ration of carbo-hydrate 40, protein 110 and fat 200 grammes. His morning blood-sugar was 0.12%.

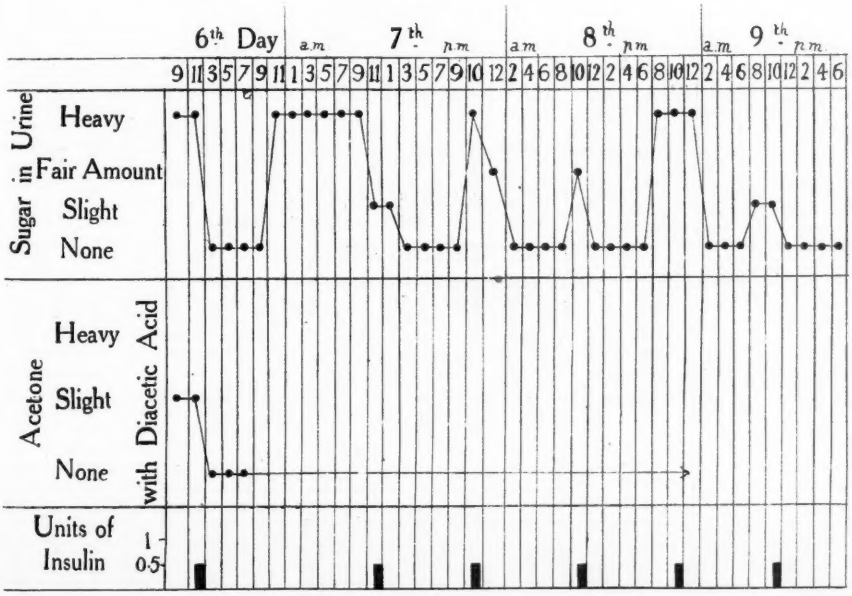
In February he had a severe attack of lobar pneumonia from which he recovered, but with so reduced a carbo-hydrate tolerance that he manifested glycosuria with a daily ration of twenty grammes of carbo-hydrate. On April 3, he was treated with two "rabbit units" of "Insulin" daily and this amount has been continued ever since. He now takes carbo-hydrate 40, protein 100 and fat 120 grammes, with only an occasional trace of glucose in the urine and no acetonuria. His weight has increased from sixty-two kilograms to sixty-six kilograms. He tells me in a letter that he feels "considerably better and with more energy" (June 11, 1923). His blood-sugar two hours after his mid-day meal is 0.17%. He is able to do light work managing his estate.

TABLE II.

Date.	"Insulin." "Rabbit Units."	Diet. Carbo-hydrate. Grammes.	Protein. Grammes.	Fat. Grammes.	Calories.	Urine. Volume. c.cm.	Sugar.	Acetone.
1923.								
March 29	None	41	104	200		1,164	6.8%	—
March 30	None	45	102	200	2,389	2,016	Heavy	—
March 31	None	20	50	10	370	2,442	Heavy	None
April 1	2	20	50	10	370	1,548	Slight	Slight
April 2	2	20	70	50	793	1,576	None	Trace
April 3	2	30	75	75	1,315	1,832	Trace	None
April 4	2	30	76	100	1,315	2,045	None	None
April 5	2	40	100	100	1,477	2,329	None	None
April 6	2	40	100	120	1,627	2,386	None	None

Case II.—R.M., aged forty years.

GRAPH I.



Case III.—D.H., a little boy of six years, was sent to me by Dr. A. M. Cudmore in October, 1919. His grandmother, her sister and his first cousin were diabetic. When first seen by me he was suffering from severe diabetes of three months' duration. His urine contained 7.5% of glucose. There was much ketonuria. His mental state was apathetic, his skin was dry and his whole aspect suggested rapid wasting and threatened coma. Under treatment his acidosis disappeared and his urine remained sugar-free with a daily carbo-hydrate intake of sixty grammes. For three years he was kept "sugar-free" though his carbo-hydrate tolerance decreased to forty grammes daily. In January, 1923, his toler-

ance had fallen so much that with only five grammes of carbo-hydrate daily he was excreting twenty-two grammes of glucose in the urine, whilst ketones were constantly present. Graph I. (page 195) shows the effect of "Insulin" on the glycosuria. Specimens of urine were passed every two hours during its administration and were at once examined for glucose.

Dr. Ray Hone has prepared Table III., which shows the biochemical changes produced by "Insulin" with varying carbo-hydrate intake. This boy is now having a daily ration of carbo-hydrate 18 grammes, protein 50 grammes and fat 60 grammes (850 calories). He receives two "rabbit units" of "Insulin" daily. His early morning blood-sugar is 0.12% and he has no glycosuria. Acetone is occasionally present.

This child has shown great general improvement. He volunteers the remark that he feels "much less tired" when he is having "Insulin" and he is quite stoical about the hypodermic injections. He has gained 1.5 kilograms in weight since "Insulin" has been administered. Unfortunately his natural tolerance for carbo-hydrate is so low that he is really being kept alive by "Insulin." Until quite recently the supply of "Insulin" was inconstant, which accounts for some of the days of omission shown in the charts. On these occasions it was not deemed wise

TABLE III.

Date.	Carbo-hydrate intake during previous 24 hours. Grammes.	"Insulin" given day previous.	Alveolar CO_2 tension day previous.	Blood sugar on day previous.	Urine Collected During Previous Twenty-four Hours. Volume. c.cm.	Total Acidity.	Total Nitrogen. Milligrammes per 100 c.cm.	Ammonia Nitrogen. Milligrammes per 100 c.cm.	Total Sugar. Grammes.	Dextrose / Nitrogen.	Ammonia / Nitrogen × 100
1923.											
Jan. 5 ..	5.0	None	20	0.21%	830	38.2	1300	84	22.0	1.6	6.5
Jan. 6 ..	5.0	None	20	0.2%	850						
Jan. 7 ..	5.0	½ unit 10.30 a.m.	30	0.11%	1835	22.0	930	45	27.5	1.3	5.7
Jan. 8 ..	5.0	½ unit 10 a.m. ½ unit 10 p.m.	25	0.14%	2360	20.0	887	34	8.7	0.18	2.4
Jan. 9 ..	11.0	½ unit 10 a.m. ½ unit 10 p.m.	25		2110	22.0	960	35	27.4	0.81	3.7
Jan. 10 ..	17.0	½ unit 10 a.m. ½ unit 10 p.m.	25		2330	20.0	770	28	32.6	0.88	3.6
Jan. 11 ..	17.0	½ unit 10 a.m. ½ unit 10 p.m.	25		1710	20.0	1085	42	15.0	—	3.8
Jan. 12 ..	17.0	½ unit 10 a.m. ½ unit 10 p.m.	25		2090	21.0	934	38	11.0	—	4.0
May 15 ..	17.75	1 unit 8 a.m.	25 at 12 noon	0.12%	1640	27.9	918	70	35.26	1.2	7.6
May 16 ..	17.75	1 unit 8 a.m.	35 at 10 a.m. 30 at noon	0.17%	1375	28.9	984	203	39.73	1.6	20.6
May 17 ..	17.75	1 unit 8 a.m.	25 at noon		1685	25.9	979	77	37.8	1.2	7.8
May 18 ..	17.75	None	20 at noon	0.22%	1899	34.7	964	115	77.8	3.3	11.9
May 19 ..	17.75	None	20 at noon		1200	50.0	1090	196	39.6	1.7	18.0
May 21 ..	7.75	None	20 at noon		875	41.0	1010	153	32.3	1.6	15.4

Case III.—D.H., aged ten years.

to attempt to keep the blood-sugar within normal limits by deprivation on account of the hunger and misery that the child would experience from too great reduction. Now that he receives "Insulin" in adequate dosage he really has quite a liberal allowance of food.

Case IV.—H.H., a female, age fifteen years, whose grandmother has diabetes, has had symptoms since October, 1921. In January, 1922, she excreted eighty grammes of glucose daily on an ordinary mixed diet. It was then found that she could take a diet of carbo-hydrate 41 grammes, protein 72 grammes and fat 172 grammes, without glycosuria and with a normal blood-sugar. Her weight was thirty-two kilograms. In November, 1922, her blood-sugar one hour after breakfast (carbo-hydrate ten grammes) was 0.13%. Her urine was free from sugar on carbo-hydrate 30 grammes, protein 80 grammes and fat 110 grammes *per diem*, but contained acetone. She had an alveolar abscess which probably lowered her tolerance. Her weight was forty-one kilograms. From this date her tolerance gradually sank, so that she had constant glycosuria and acetonuria on a daily allowance of twenty-five grammes of carbo-hydrate in March, 1923. Her weight fell to thirty-nine kilograms. "Insulin" was then given in doses of one "rabbit unit" twice daily. Her urine still contains sugar several times a day with twenty grammes of carbo-hydrate daily intake. The acidosis is much less. Her blood-sugar is usually below 0.15%. She has gained two kilograms in weight and feels much better. I shall increase her dose of "Insulin" now that we can obtain more.

but was still wasting. His urine was loaded with sugar. His toe had begun to heal, but the wounds were discharging freely. On June 13 his diet comprised fifty grammes of carbo-hydrate, one hundred grammes of protein and one hundred grammes of fat. His blood-sugar was 0.4%. One "rabbit unit" of "Insulin" was given before breakfast and tea on June 13, 14, 15 and 16. On June 16 the last dose of "Insulin" was given. The urine contained a trace of sugar. On June 17 and 18 no sugar was present in the urine. He felt and looked better. The discharge from the wounds was less.

On June 20 the sugar reappeared and persisted until June 25. On this day the blood-sugar was 0.26%. Two "rabbit units" of "Insulin" were given before breakfast and one "rabbit unit" before tea. His diet contained thirty grammes of carbo-hydrate, eighty grammes of protein and one hundred grammes of fat. He also received eighty cubic centimetres of brandy. On June 26 his urine contained no sugar nor ketones. His blood-sugar was 0.11%. On July 2 he was taking forty grammes of carbo-hydrate, one hundred grammes of protein and one hundred grammes of fat, equivalent to 1,600 calories. No "Insulin" was given on July 7. His diet contained twenty grammes of carbo-hydrate, fifty grammes of protein and fifty grammes of fat. There was a trace of sugar in his urine. On July 9 there was no sugar in the urine.

From July 10 to 15 he was given one "rabbit unit" of "Insulin" while taking thirty grammes of carbo-hydrate,

TABLE IV.

Date.	Diet.			"Insulin."	Urine.	
	Carbo-hydrate. Grammes.	Protein. Grammes.	Fat. Grammes.	Units.	Volume. c.cm.	Glucose. Grammes.
July 6, 1923	20	100	130	3	1,650	46.2
July 18, 1923	20	100	130	None	2,340	81.9

Case IV.—H.H., aged fifteen years, showing the effect of three "rabbit units" of "Insulin" on glycosuria.

Diabetic Gangrene with Severe Sepsis.

Case V.—A man, Dr. Messent's patient, *ætatis* sixty-three, a clerk, has severe diabetes. He has lost thirteen to nineteen kilograms in eighteen months. On April 4, 1923, his urine had a specific gravity of 1,040 and contained "heavy" sugar. His left great toe was becoming gangrenous. This was incised freely. On April 13 he had become "sugar-free" after starvation and was now taking fifteen grammes of carbo-hydrate, thirty-four grammes of protein and eighteen grammes of fat. On April 14 cellulitis began in left popliteal region. This rapidly spread. The patient's general condition became extremely grave. On April 18 much acidosis was present and a low muttering delirium set in. Carbo-hydrate was given freely and treatment for acidosis carried out. On April 21 and 22 0.5 "rabbit unit" of "Insulin" was injected night and morning. The result was not noticeable. On April 23 he was very drowsy, refused food and drink. Saline and glucose solution was injected into the rectum every four hours. On April 25 his condition was much better. The urine contained "heavy" sugar and diacetic acid. From April 26 to May 10 his diet consisted of fifteen grammes of carbo-hydrate, fifty-two grammes of protein and thirty-nine grammes of fat. The urine only occasionally contained a trace of sugar, but the cellulitis extended. Pus discharged freely from incisions which were made at intervals from *tuber ischii* above to the ankle below. Large sloughs of the hamstring muscles separated. The general condition rapidly deteriorated; wasting was severe and progressive. The mind wandered at night. The urine at times contained diacetic acid. As the patient appeared to be dying from exhaustion and sepsis, the diet was increased; bread and biscuits were allowed when desired.

Since the increase of diet he felt much better on May 20, eighty grammes of protein and one hundred grammes of fat. A trace of sugar was occasionally present in the urine. On July 16 to 20 no "Insulin" was given. His diet consisted of fifteen grammes of carbo-hydrate, sixty grammes of protein and eighty grammes of fat, yielding 1,095 calories. His urine was usually sugar-free, but occasionally contained a "trace."

The general condition improved steadily since the last course of "Insulin" was started. The patient feels and looks stronger and has put on flesh. The pulse is slow, strong and regular. The tongue is moist. The discharge from the wounds has ceased and they have healed with the exception of a small sinus in the popliteal space. Before the "Insulin" was given, the wounds were discharging large amounts of thick pus and showed no tendency to heal. The patient looked as if he would die in a few days. Now he is beginning to walk and says that he feels as well as he has for years.

Threatened Coma.

Two cases of this kind have occurred, but owing to the scanty supply of "Insulin" at the moment we were unable to use it freely.

Case VI.—A girl, aged twenty, had suffered from severe diabetes for five months. When seen she was almost unconscious, awakening only to answer questions. Wasting was extreme. Her respiration was panting and the breath reeked of acetone.

Graph II., prepared by Dr. West, shows the progress of the condition under treatment. The extreme depression of the alveolar carbon dioxide is to be noted and its gradual rise under treatment. Unfortunately symptoms of broncho-

Case III.—D.H., aged ten years.

pneumonia appeared and at about the same time our supply of "Insulin" was exhausted. She died rather from terminal pneumonia and exhaustion than from coma.

Case VII.—A.S., a man, aged twenty-three, had diabetes of two months' duration. On admission, he was in a state of threatened coma. His alveolar carbon dioxide was fifteen millimetres of mercury. His blood-sugar was 0.33%. Twenty-four hours' urine amounted to 6,600 cubic centimetres with 4.8% glucose on an intake of 75 grammes of carbo-hydrate.

Graph III., prepared by Dr. West, shows the treatment adopted and the result. On the fifth day one "rabbit unit" of "Insulin" was injected and the alveolar carbon dioxide which had been stationary at twenty-five millimetres of mercury for twenty-four hours, rose in the next twenty-four hours to thirty-five millimetres of mercury.

Neither of these cases can be regarded as crucial examples of the action of "Insulin" in severe acidosis. The first patient was apparently too exhausted as well as being intensely acidotic; the second was already improving when the "Insulin" was given.

Other patients have been treated in the Adelaide Hospital and in the Adelaide Children's Hospital, but they resemble those already described in regard to the action of "Insulin."

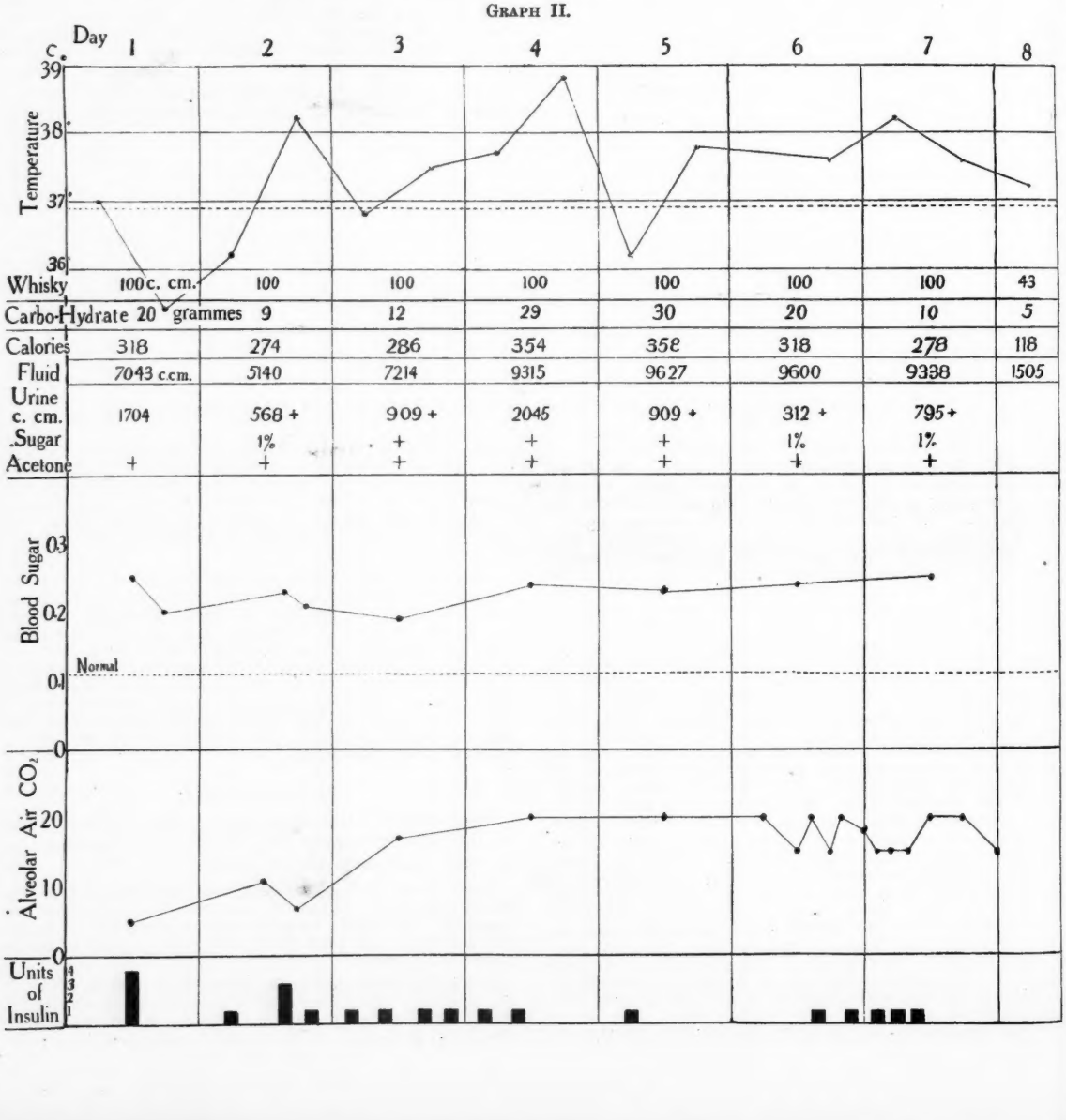
Conclusions.

From a consideration of the experience recounted above and the recorded findings of others the following conclusions have been formed.

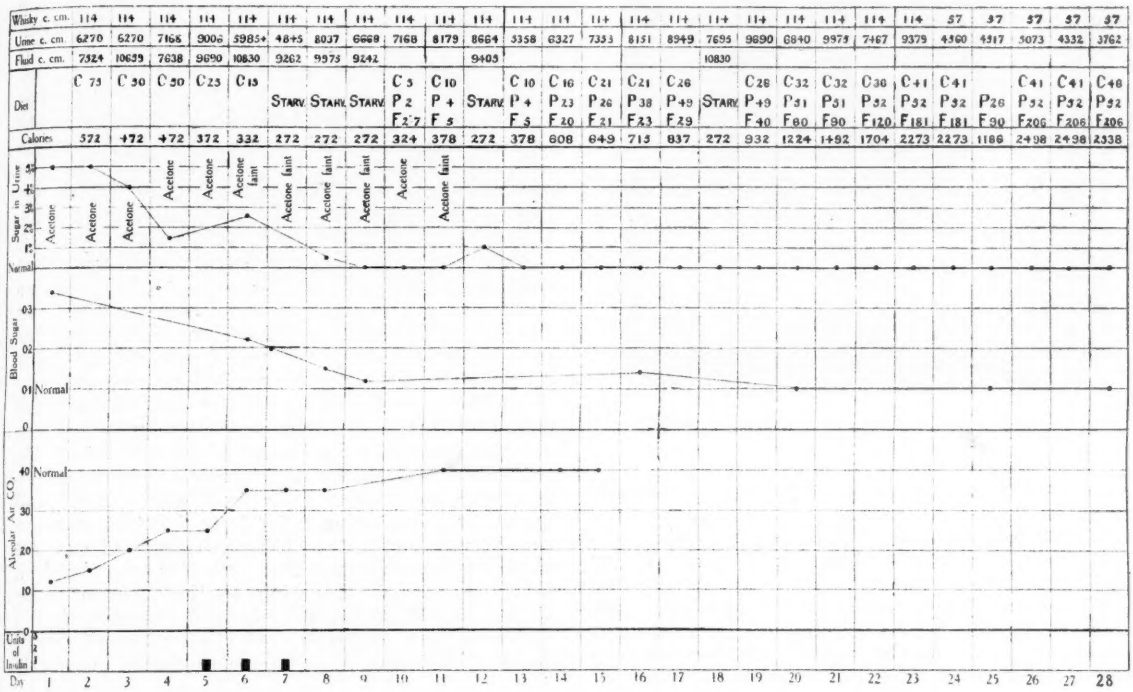
1. In an individual patient, a definite dose of "Insulin" will metabolize a definite amount of carbohydrate.

2. In a patient suffering from severe diabetes this fraction may represent the difference between exhaustion and a fair measure of health.

GRAPH II.



GRAPH III.



3. The action of "Insulin" lasts for a few hours (about ten) after its injection.

4. It has no permanent action.

5. Diet must be as carefully controlled during administration as it is without.

6. Its use is indicated in: (*a*) Severe diabetes with a carbo-hydrate tolerance below thirty grammes *per diem*, (*b*) cases of acidosis where coma threatens, (*c*) diabetes before surgical operation and anæsthesia, (*d*) diabetes when some intercurrent complication, septic, infective or other, has lowered tolerance or increased acidity or both.

7. Its use should be guarded by careful diagnosis and estimations of blood-sugar.

8. The dose must be regulated by carbo-hydrate tolerance and weight of the patient and the percentage of blood-sugar.

9. The usual dose used by use was two to three centimetres ("rabbit units") daily, but this has been usually too small and I believe in most cases should be increased to four (old) units daily.

10. The principal meals of carbo-hydrate should be given half an hour after the administration of "Insulin."

11. "Insulin" is not indicated in the treatment of diabetes when diet will keep the patient sugar-free, without acidosis and with sufficient calories.

I desire to express my indebtedness to Professor Brailsford Robertson and his assistants, Mr. Anderson and Miss Cheadle, who have rendered "Insulin" available for our use. I have received much assistance from my house physicians, Dr. West (of the Adelaide Hospital) and Dr. Britten Jones (of the Children's Hospital) and also from several of my clinical clerks. Dr. Ray Hone, working in Professor Robertson's laboratory, has carried out the metabolic studies shown in Tables I. and IV.

SOME DEFECTS IN THE FOOT THAT CAUSE DISABILITY.[1]

BY W. KENT HUGHES, M.B. (London),
Honorary Surgeon, Children's Hospital, Melbourne.

Diminished Dorsal Flexion.

THE first defect to which I wish to draw attention is diminished dorsal flexion. This has been described in its severest form under *talipes equinus*, in its less severe as a right-angled talipes or non-deforming club-foot. In 1885, Shaffer brought the subject forward in *The New York Medical Record* under the latter synonym. There are, however, slighter forms of the defect which give rise to serious troubles. I will take first a foot which cannot be dorsi-flexed beyond a right angle with the leg (see Figure I.). It is apparently well formed and that is why Shaffer called it non-deforming club-foot. In order to determine the existence of such defect it is necessary to keep the leg well extended in order to determine the extent of the shortening of the *tendo Achillis*. In a normal foot the dorsal flexion should proceed 20°

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 4, 1923.

FIGURE I.—METHOD OF TESTING FOR DORSAL FLEXION OF THE FOOT.

beyond a right angle. A very large number of people have much less than this and a great many have only half this amount (see Figure II.).

Some have ascribed this condition to faulty methods of walking and improper boots. Personally I believe it is in most cases a congenital defect and that instead of *talipes equinus* being the rarest of all congenital conditions, it is by far the commonest, that is to say if we class all cases of diminished dorsal flexion under this heading.

A *tendo Achillis* that is short from birth, may remain relatively short as the child grows or the shortening may become more pronounced and produce a gross form of *talipes equinus* or *equino-varus cum cavo*. Such a type is well known under what is called hereditary or familial *talipes equino-varus*. For example, may I quote the cases that I showed at the last meeting of the Pædiatric Society? A man named L. came to me fourteen years ago, at the age of twenty-eight, with gross *talipes equino-varus*. At fourteen years of age, he began to have trouble with his feet and the condition increased until he was quite crippled. He married and has four children. The two elder began between six and seven years of age to be clumsy on their feet and when brought to Children's Hospital at eight and ten, had severe *talipes equino-varus*. A younger child, aged five years, has right-angled talipes on one side and dorsal flexion limited to 80° on the other. He has several relatives similarly affected. During the past thirty years, I have had four cases of families with a similar history. They are in every way similar to the cases that arise from anterior polio-myelitis excepting, of course, there is an absence of paralysis and reaction of degeneration and the limbs, though not well developed, are quite strong. On the other hand we have the cases in which the dorsal flexion is only limited to about 80° or 78° and does not proceed any further. These are the types that are overlooked when foot disability ensues. Even right-angled talipes is frequently missed because the foot looks normal and the patient obtains no relief from flat foot or vague muscular pains in leg or back because the source of the trouble still remains. Shaffer was the first, I believe, to call attention to the frequent association of scoliosis with diminished dorsal flexion.

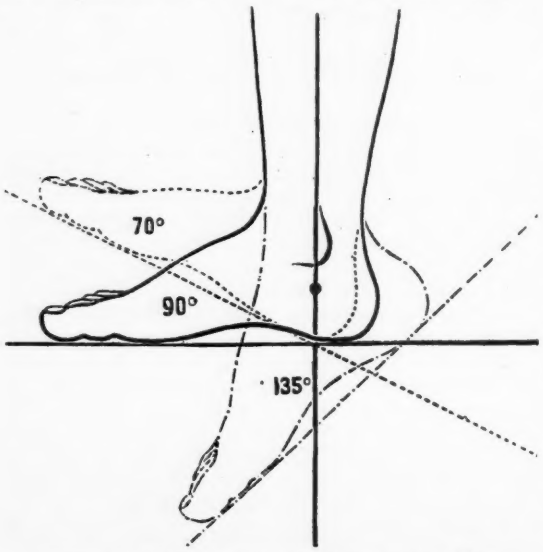


FIGURE II.—RANGE OF DORSAL FLEXION OF THE FOOT.

The earliest indication of diminution of dorsal flexion is evidenced (i.) by clumsiness, especially in getting over fences or walking upstairs; (ii.) by easily getting tired on walking uphill, often with swelling of tissues of the foot and vague pains; (iii.) by weak ankles; (iv.) by sudden giving way of the foot when walking, especially on uneven surfaces, the patient frequently falling over.

With the exception of *"valgus"* ankles diminution of dorsal flexion seldom gives rise to any appreciable disability until after adolescence has begun. Up to that time the child may be clumsy on its feet or may be behind its fellows in agility; from then on, however, increasing disability is manifested. The condition may progress and so produce a definite equinus position. In this connexion it is noteworthy that the Swiss mountain guides have an increase of dorsal flexion to 10° and 15° beyond the normal. In the slightest forms the patient gets easily tired, especially when walking up hill. The majority of the patients who have come under my notice, have come for treatment between eighteen and twenty-five years of age, but some have proceeded to well on in middle life and a few have passed the fifth decade before seeking relief.

Very many cases of flat foot arise from this defect. A patient finds that he cannot walk freely with his foot directed forwards, so he turns his foot outwards to get freer action. This helps to break down the inner border and flat foot results.

Large masses of thickened epidermis are often found in these cases under the ball of the big toe and often across the sole of the foot at this level. Nearly every case of a contracted *tendo Achillis* also has a well formed *cavus*, though the converse is not true. I have seen many cases of *cavus* without any shortening of the *tendo Achillis*.

FIGURE III.—HALLUX FLEXUS OF THE DISTAL JOINT.

Another condition frequently associated is dorsal flexion of the toes; as the big toe is so much the most prominent, many observers have only noted the presence of *hallux flexus* and have ignored the condition of the smaller toes (see Figure III.).

Talipes cavus especially when not associated with *equinus* is generally overlooked. It is often ascribed to the wearing of short boots, but I think that, however much faulty footwear may help, there is a congenital condition at the outset in most if not in all cases. Like a short *tendo Achillis*, contracted muscles of the sole may grow relatively with the rest of the foot or they may fall behind and so increase the *cavus* condition. As in *cavus* following upon infantile paralysis this deformity is expressed by a plantar flexion of the foot anterior to the medio-tarsal joint.

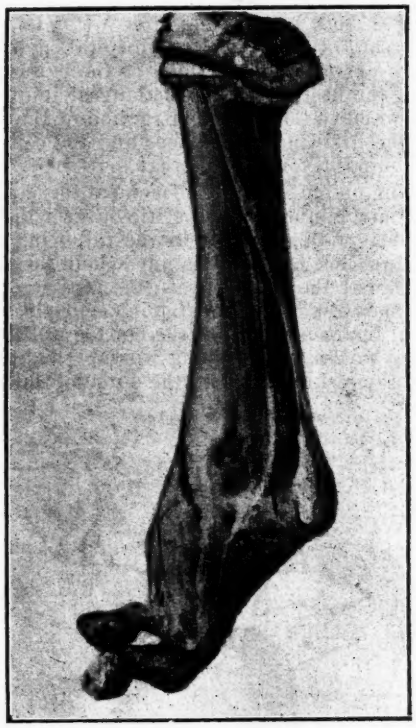


FIGURE IV.—TALIPES CAVUS.

Treatment of *talipes cavus* whether by extirpation of the astragalus or by shortening of dorsal flexors has not been satisfactory. For many years, I have followed the plan of dealing with the fibrous elements of the foot in a fairly drastic manner. An incision is made through the skin along the inner border of the foot from below the tip of the malleolus to the metatarso-phalangeal joint; through this incision the superficial plantar fascia is thoroughly freed from the deeper structures right across the foot to its outer border; if necessary it is then divided transversely, but often this will be unnecessary. Then the *abductor hallucis* is freed from the metatarsal bone along its whole length and drawn outwards with a speculum and flat elevators. The operator then proceeds across the foot, keeping close to the bones, cutting all the strongly contracted bands of fascia coming down to be attached to the tarsal ligaments. The long and short plantar ligaments are also divided, care being taken not to injure the *peroneus longus* tendon (see Figure IV.).

In advanced cases it may be necessary to proceed carefully along the inner border, cutting the fascial origin of the *abductor hallucis* and even opening up the joints between the scaphoid and its neighbours. In all but the worst cases the *cavus* is now overcome and the foot unfolded at the medio-tarsal joint and the metatarsal bone brought into its proper relation with the posterior bones of the foot. If there is any *equinus* present, the *tendo Achillis* is lengthened.

In the worst cases the incision must be continued on to the heel, the skin dissected loose from the structures passing from the leg to the foot; a deep cut is now made round the side and anterior edge of the *os calcis*.

Hæmorrhage may be troublesome if there is a large internal plantar artery and occasionally the artery to the metatarsal phalangeal joint of the fifth digit may be difficult to seize with forceps.

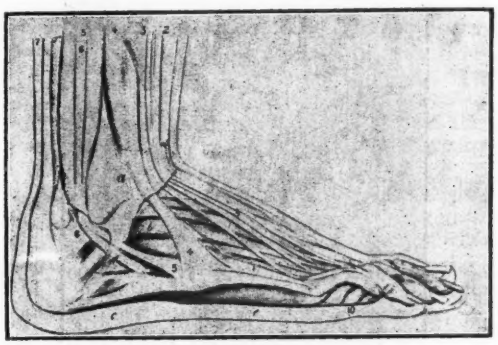


FIGURE V.—ANATOMY OF TENDONS OF FOOT TO SHOW THE RATIONAL TREATMENT OF PES CAVUS.

The patient I am showing you tonight with a perfectly flat foot is an example of what can be done by the operation I have described for *pes cavus*. His is an example of *pes cavus* resulting from polio-

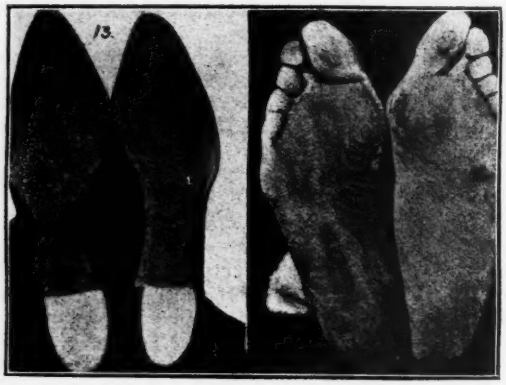


FIGURE VI.—PRODUCTION OF HALLUX VALGUS BY ILL-FITTING BOOTS.

myelitis of long standing and illustrates the most extreme type.

Hallux Valgus and Bunions.

Hallux valgus is certainly produced by faulty footwear, both socks and boots, but as everybody practically wears both, I have long taught that some predisposing condition is present in those who suffer from pronounced *hallus valgus*. An adult with a perfectly straight big toe is practically not to be found in an English-speaking community. Yet those who suffer from *hallux valgus*, only form

FIGURE VII.—FAULTY FOOT-WEAR PRODUCING HALLUX VALGUS.

FIGURE VIII.—PRODUCTION OF HALLUX VALGUS BY ILL-FITTING BOOTS.

a very small percentage. Again in those who suffer, there is, as a rule, a great difference in the condition existing in both feet (see Figures VI., VII., VIII.).

Anderson, of St. Thomas's Hospital, pointed out long ago extreme contractions of the external ligament of the first metatarso-phalangeal joint. Added to this we have the great contraction of the skin between the first and second toes and the contraction of the dorsal flexors of the great toe (see Figure IX.).

Leaving on one side for the moment those who have an inflamed bunion or periostitic outgrowth, we can easily deal with all cases in the following manner (see Figure X.). Get an assistant to hold the great toe inwards; cut the skin between the toes freely down to the metatarso-phalangeal joint; cut through the external ligament and pull the big toe well inwards. We now have a large gaping wound which must be filled in by a skin flap from the dorsum, the size and

FIG. 14.

FIGURE IX.—HALLUX VALGUS.

FIGURE X.—INFLAMED BUNION.

extent of the same depending upon the gap that is made. A narrow bandage drawing the big toe inwards and a good-sized plug of dressing between the toes is all that is required. At the end of a week or so make the patient walk a little, increasing his exertions each day.

In cases with an inflamed bursa this must be dissected out and if there are periostitic outgrowths, these must be chiselled off. No splints are required and with the exception of a digitated sock and proper boot no special after treatment need be insisted upon.

As to ætiology, I hold that in cases requiring treatment there is either a congenital shortening of the dorsal flexors or an inherent tendency in the external lateral ligament to contract.

Hammer Toe.

All writers on hammer toe deal solely with the first interphalangeal joint. They ignore the contracted ligament on the dorsal surface of the metatarso-phalangeal joint and many overlook the contracted dorsal flexor. I always deal with these two structures first and obtain full plantar flexion at this joint. It makes the dorsal flexion at the interphalangeal joint so much easier to obtain.

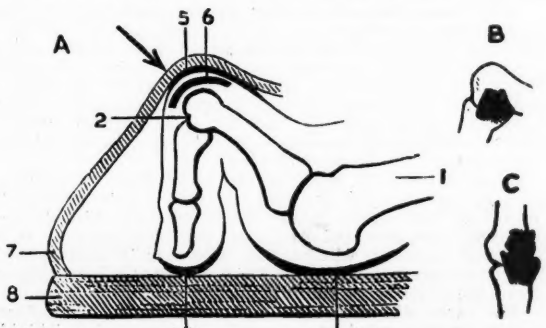


FIGURE XI.—HAMMER TOE.

Amputation of the toe is a surgical disaster and must be avoided at all cost; a weakened foot is the inevitable result with the certainty of a flat foot ensuing.

Corns.

Corns are generally considered beneath the dignity of a surgeon's notice and so people are driven to seek the advice of pedicurists and chiropodists. It would be difficult to estimate the sum total of the pain and disability caused by corns, to say nothing of the enormous amount of time and money that is spent in obtaining temporary relief.

Get a proper pair of boots and your corn will disappear is generally the beginning and the ending of the surgeon's advice. Now that is not true, for there is often an underlying anatomical condition that will produce corns in any boot with an "upper" and that is the very common dorsal flexion of the toe at the metatarso-phalangeal joint. The little toe is, perhaps, the commonest digit to suffer, for here we have intermittent pressure from the outside as well as intermittent pressure on the dorsal surface.

Corns may be grouped in three classes: (i.) Corns on the dorsal surface of the interphalangeal joints; (ii.) soft corns between the toes; (iii.) corneous masses on the sole of the foot.

Corns of the second group undoubtedly are due solely to improper footwear in both socks and boots. If a digitated sock can be obtained and a well formed boot be ordered, there should be no difficulty in banishing the corn. Soaking the part in alum water and dusting the surface with equal parts of boracic powder and alum will be found useful. If a digitated sock cannot be obtained, get a good wide sock and place pledgets of cotton wool between the affected toes.

For the first and third groups it is often quite useless simply to order surgical boots and to use one of the many corn solvents. We must in the first obtain a freely movable toe with capacity of full plantar flexion at the metatarso-phalangeal joint; that is to say nearly every person with corns on the dorsal surface of the second, third and fourth digits will be found to have some degree of hammer toe, possibly without plantar flexion of the toe at the first interphalangeal joint being strikingly evident.

In the third group, as mentioned earlier in the paper, there is nearly always to be found some diminution of dorsal flexion at the medio-tarsal or ankle-joint. When this is absent, a corn on the sole can only be produced by an uneven inner sole of the boot.

I find that many of the corn solvents are too strong and produce irritation of the adjacent skin. I think the safer plan is to use varying strengths of an ointment containing salicylic and carbolic acids. Begin with 1.14% (five grains to the ounce) and use if necessary up to 4.56 % (twenty grains to the ounce) of each ingredient. I much prefer lard to vaseline; as Sir Dyce Duckworth used to say: "Vaseline is very good for machinery."

Reviews.

"PHYSICS AND BIOLOGY OF RADIATION THERAPY."

Although Friedrich and Kroenig's work on "The Principles of the Physics and Biology of Radiation Therapy" was published in German in 1918, the English translation was only authorized after the visit of Henry Schmidt to Freiburg, and it is to him that we owe the present translation.[1] Divided into two portions, one dealing with the physical and the other with the biological problems, the great value of this work lies in the fact that it represents the work of a physicist and a clinician attempting in collaboration to solve the difficulties of radiation therapy. Its completeness is well shown by the care exercised in ascertaining the most suitable and reliable instrument for measuring the intensity of radiation. The ultimate choice fell on the iontoquantimeter, the special construction of which is described in detail together with the experimental data obtained in testing it. This information is invaluable for those contemplating the purchase of an iontoquantimeter.

The different influences affecting the percentage of the depth dose were examined carefully, once the reliability of the iontoquantimeter had been ascertained. Use was made of the water phantom, as the absorption coefficient for water was found to be almost identical with that of the human tissues for the radiation employed. That portion of the work dealing with the biological problems is no less accurate and complete. Using frog spawn as objects for biological tests, such diverse subjects as the action of diathermy and of heat on the biological effect of X-rays were investigated. Similar work was carried out regarding the γ radiation of radium and mesothorium. The main finding was that the class of tissue irradiated affected the degree of response more than any outside influence. The authors attempted to reduce this finding to figures and placed the dose required to produce an erythema at 170*e* ("*e*" representing the quantity of rays which transports in one cubic centimetre of air by ionization the quantity of electricity of an electrostatic unit with a saturated current). The ovarian dose was placed at 33*e* and the carcinoma dose at 150*e*. Experience has shown that these figures must be looked upon as merely a working basis for the determination of the optimism dose for each patient.

Not the least valuable portion of the book is the appendix which records experiments made since 1918 and serves to bring the book up-to-date. It deals especially with the influences of the focal-skin distance upon the percentage of the depth dose and with the use of secondary radiation from solids and solutions. The conclusion reached is that secondary radiation is of doubtful value in therapy with the hardness of rays in use at present.

A series of coloured plates illustrating the various experimental results when the skin was used as a biological test object, are gathered together at the end of the book and are of inestimable value to those far removed from their fellow workers.

A HANDBOOK OF PRACTICAL BIO-CHEMISTRY.

THE authors of a laboratory handbook of bio-chemistry,[2] P. C. Raiment and G. L. Peskett, have produced a pleasing well got up book, clearly printed and with the contents carefully arranged so that quick reference can be made to any desired test. The book is intended for the use of junior students. The authors have followed the plan of giving a short résumé of the theoretical work on which the tests are based at the commencement of each section. They hope that this arrangement will enable the student to perform the tests more intelligently. The subject matter of the book, however, is sadly disappointing. It is just like the hundreds of other textbooks of practical bio-chemistry that were produced in days gone by when the subject was yet in its infancy. To choose two examples, subjects such as the enumeration of the red corpuscles and the microscopical detection of pus in urine sound out of place in a modern bio-chemcial handbook and yet they are included in this. Quantitative tests are scarcely ever given. In fact, on examining this volume, we can see, in view of the numerous, good, up-to-date practical manuals that exist nowadays, little reason for its production.

[1] "The Principles of Physics and Biology of Radiation Therapy," by Dr. Bernhard Kroenig and Dr. Walter Friedrich, of the University of Freiburg im Breisgau, with an appendix by Dr. Henry Schmitz, Chicago; 1922. London: William Heinemann (Medical Books), Limited; Demy 4to., pp. 271, with 86 figures in the text and 20 coloured and 11 black and white plates and 32 tables. Price: 42s. net.

[2] "A Laboratory Handbook of Bio-chemistry," by P. C. Raiment, B.A., M.R.C.S., L.R.C.P., and G. L. Peskett, B.A. Edwin Arnold & Co., London, 1922.

Analytical Department.

"PEPSODENT TOOTH PASTE."

"PEPSODENT TOOTH PASTE" is manufactured by The Pepsodent Company (Australia), Limited, at 139, Clarence Street, Sydney. Our inspector has visited the factory and reports that it is well lighted, well ventilated and very clean. The girls employed in tending to the filling machines and the packing wear white aprons and caps and strict attention appears to be paid throughout to cleanliness.

"Pepsodent" contains calcium phosphate, calcium sulphate, pepsin, gums, hydrochloric acid, flavouring and antiseptic agents, glycerol and water. The calcium phosphate, calcium sulphate, pepsin and gums are obtained from America in paper-lined barrels. They are weighed, mixed and placed in a machine which brushes the powders through a very fine brass sieve and thoroughly mixes the substances together. Glycerol, hydrochloric acid, water and alcoholic solutions of eucalyptol, menthol and oil of peppermint are mixed together and placed in a glass-lined tank. The sieved powders are placed in a mixing machine and the fluid mixture run in at a steady rate while the barrel and blades of the machine are rotating. This takes about three-quarters of an hour. The churn and blades are then rotated at a greater speed for about one and three-quarters of an hour until finally there is a homogeneous mixture of the solid and liquid constituents. The paste is then conveyed to the hopper of a filling machine; the tin tubes of paste are filled automatically and the bottoms sealed and clipped automatically. The churns and hoppers are washed with boiling water as soon as a batch of paste is finished and tubed. The process of manufacture leaves nothing to be desired.

The individual ingredients were examined and found to be of the highest quality, the pepsin being a particularly fine sample.

Analysis of a sample of the paste obtained at the factory and of one purchased outside gave:

	Factory Sample.	Purchased Sample.
Hydrochloric acid ..	0.164%	0.158%
Ash	59.96 %	57.89 %

The ash consisted of almost equal parts by weight of calcium sulphate and calcium phosphate.

A little of the paste was placed on a plate of gelatine jelly and after a few minutes washed off. The gelatine was distinctly etched, showing that the pepsin was active; both samples gave the same result. Some of the paste was mixed with 0.2% hydrochloric acid and a small piece of fibrin added and the mixture incubated at 37° C.; the fibrin was largely dissolved.

A microscopical examination of the paste showed that the calcium phosphate was in the form of a fine powder, but the calcium sulphate was present as small, sharp crystals. Rubbed between two plates of glass the paste did not produce any scratches visible under the microscope and the crystals broke into small fragments. When used as a tooth paste "Pepsodent" feels decidedly gritty; this is due to the presence of the crystals of calcium sulphate and the absence of soap.

It is claimed by the manufacturers that "Pepsodent" attacks and removes the film on teeth. This it may do partly by the cutting action of the calcium sulphate and calcium phosphate and partly by pepsin digestion. It is doubtful, however, whether the pepsin is in contact with the teeth together with acid for a sufficiently long time to have much effect in this direction.

For those who desire an acid tooth paste, "Pepsodent" can be recommended as a carefully compounded and highly efficient product.

The Medical Journal of Australia

SATURDAY, AUGUST 25, 1923.

Health and the Worker.

IN March of this year we drew attention to the importance of what may be called the industrial hygiene movement from the point of view of its economic value. It is well at the present time to consider the question from another aspect. The branch of the Commonwealth Department of Health which is concerned with industrial hygiene, has been doing its work steadily and quietly, making an impression here, starting a new centre of activity there and all the time consolidating its position. As has been pointed out on several occasions this branch of the Department has no compelling power, no right to force its conclusions or its methods on any one. It works by persuasion. It is probable that in these circumstances any advance that is made, will be the means of laying a surer and more lasting foundation on which a permanent structure may be built. Under the guidance of this branch of the Department of Health the recognition of the value of measures which safeguard the health of the workers is becoming more general. Although the work is yet in its infancy and efforts are mainly directed towards ascertaining the actual morbidity and mortality connected with various trades, it is noticeable that in an increasing number of factories and commercial houses in the capital cities as well as in the larger industrial undertakings schemes have been and are being inaugurated under which medical practitioners are made responsible for the supervision of the general health of the workers. Unfortunately the condition are such that each medical officer is dependent very much on his own devices and must make his own general working rules.

The success of undertakings of this nature has been variable. The frequent occurrence of failure in this regard has been discussed recently by Dr. René Sand, the General Secretary of the League of Red Cross Societies, in the pages of the *International Labour Review*. Dr. Sand states that in the first place health lectures, leaflets and posters have as a rule been too theoretical and pedantic and completely out of touch with the needs and mentality of the workers. The probable truth of this is evident. In Australia propaganda work has been used chiefly in connexion with Health Week in the several States. The inauguration of Health Week was largely the result of the activities of the Public Health Association of Australasia. In more than one of the States the cooperation of the medical officers of the Departments of Health was obtained. Dr. Sand's second reason for the failure of popular health propaganda is that no teaching can be wholly imposed from outside. The pupil must take an active personal share in the lessons. He quotes Michelet that, if teaching be a friendship, it presupposes a mutual interchange between teacher and taught. A class should not contain, say, one teacher and thirty pupils but thirty-one teachers and thirty-one pupils. To achieve the maximum success information by lectures, propaganda and so forth should be demanded and not imposed. Dr. Sands suggests, and this is pregnant with possibilities as far as Australia is concerned, that the interest of the members of friendly societies should be enlisted. The percentage of the population in the Commonwealth belonging to friendly societies is very large and once it has been made clear that benefit will accrue from the adoption of hygienic precautions and standards in industry, the path of progress should be easier to travel. A third reason for the failure of health propaganda efforts is the fact that attempts towards education and health are incompatible with extreme poverty. It is useless offering advice to a man who has not got the bare necessities of life. This condition does not obtain in Australia to nearly the same extent as in older parts of the globe. Dr. Sands uses this as an additional argument for obtaining the help and interest of friendly societies. The final reason for failure advanced by Dr. Sand is lack of coordination of the various efforts made. The importance of coordination cannot be overestimated. Its effectiveness is well evidenced in the work that has been done in Halifax and has been reported in the pages of this journal by Dr. Franklin Royer. It is interesting,

moreover, to note that in Europe at the Fourth International Labour Conference a resolution was submitted to the Governing Body of the International Labour Organization with a view to considering ways and means of realizing cooperation between employers, workers and the Red Cross Societies for "furthering the well-being and the health of the populations."

The duty of the members of the medical profession in Australia in regard to industrial hygiene is clear. The majority of medical practitioners should be seized with the importance of preventive as distinguished from curative medicine. The advance of preventive medicine as a definite and real thing is being steadily and surely made. Industrial hygiene is essentially concerned with the prevention of ill-health. Disease in the community must become lessened as industrial hazards are reduced and as the environment of the worker is improved. It is essential that the medical mind should approach the subject from a scientific and not from a philanthropic or humanitarian aspect. Moreover, the industrial hygienist as such knows no political party platform or faction. The duty of medical men in Australia is one of educating others. The opportunity of the general practitioner in the homes of the people is unequalled. Before he can teach others he must himself be a successful pupil. In this connexion it is well to note that opportunity is knocking at his door. The *motif* of the forthcoming Congress in November is preventive medicine. Disease in all its aspects will be looked at from this standpoint. The section of hygiene in the Pan-Pacific Science Congress also offers an opportunity for information and stimulation of interest in the many problems connected with hygiene in this part of the globe. Additional opportunities are continually being offered in the pages of this journal devoted to abstracts from current medical literature. Since the beginning of this year abstracts from various journals on the subject of hygiene have been introduced in rotation with other subjects. It is to be hoped that practitioners of medicine will seize every opportunity of equipping themselves for dealing with this problem. Determination will lead to knowledge and a wider outlook; knowledge will render accomplishment possible.

Current Comment.

MYOSITIS OSSIFICANS PROGRESSIVA.

ALTHOUGH Patin in 1692 described the condition of a patient suffering from progressive *myositis ossificans*, it was not until 1869 that the condition was accurately described by Münchmeyer. He used the term *myositis ossificans progressiva*, for he regarded it as a constitutional disease associated with inflammation of muscles and characterized by slow progress and periodic exacerbations. He described three stages in the muscular lesion: Firstly an inflammatory swelling, secondly fibrous induration with excessive growth of intermuscular and tendinous connective tissue and finally ossification. Cahen described certain histological changes in the muscles. He found that the muscle fibres lost their striation, disappeared and were replaced by fibrous tissue in which bone was laid down. Goto investigated the histology of the condition and came to the conclusion that the fibrous tissues were the primary seat of the lesion. He suggested the name *hyperplasia fascialis ossificans progressiva*. Krause and Trapper studied the radiological appearances and found rarefaction of the bones of the skeleton accompanying the growth of bone in the muscles. A congenital deformity of the great toes was decribed by Helferich as occurring with great frequency. Other observers, among them Jüngling and Stephen Paget, have also drawn attention to this. Virchow saw a close relationship between *myositis ossificans progressiva* and multiple exostoses and Pincus and Roth maintained that the periosteum played an important part in the causation of the former condition. It is well to remember that the progressive form of the disease is distinct from the circumscribed and the traumatic forms. In the traumatic form there has usually been an injury to the periosteum and this most likely has acted as the starting point of the deposition of new bone.

Dr. J. J. Nutt has recently reported a case of *myositis ossificans progressiva* occurring in a boy ten years of age.[1] The condition appeared at the age of eighteen months, six months after a fall. He did not become much worse till he was five years of age. After that time the disease gradually progressed. It is not necessary to describe the condition of the patient in detail. It is sufficient to indicate that bone formation had taken place in the majority of muscles above the knees. No microdactylia was present. Blood analysis showed a decrease in the calcium content. Dr. Nott also states that after exhaustive studies of the metabolism nothing abnormal was found. The excretion of calcium was unaffected. Unfortunately he gives no details of his work in this direction. He refers to the work of Austin in regard to the calcium metabolism of a patient who suffered from *myositis ossificans progressiva*. Austin failed to find any alteration in the calcium metalbolism. Austin stated that the patient seemed to offer a perfectly normal metabolism which consisted in a calcium

[1] *The Journal of Bone and Joint Surgery*, April 1, 1923.

equilibrium on a mixed diet of sufficient amount, a loss on an insufficient amount of a mixed diet and a loss on an ample diet of foods poor in calcium.

Dr. Nott records the summaries of fourteen case histories of instances of the disease reported by other observers. These with those reported in the literature bring the total of reported cases up to one hundred and thirty. He concludes his article with an extensive bibliography of one hundred and forty-four references.

THE FLOCCULATION REACTION IN SYPHILIS.

REFERENE has been made in previous issues of this journal to the test introduced by Sachs and Georgi in 1918 for the diagnosis of syphilis. These investigators observed that the globulin of the serum of syphilitics could be thrown out of solution by the addition of extracts of certain organs and a solution of cholesterin. The separation of the globulin takes places in the form of a flocculation. They devised a method of applying this test and others have used the same technique with good results. Unfortunately the test, although quite simple and easy to apply, does not yield its results at once. In 1921 Dreyer and Ward, working at Oxford under the Medical Research Council, devised a quantitative test based on this technique. They renamed it the "sigma test." The introduction of quantitative measurements involved a complication in the technique and thus deprived the innovation of its general utility. Yet another modification has been introduced. This time the technique has been simplified. Dr. A. H. Priestley sought to apply the method first described by Coca and subsequently extended by Garrow of expediting the biological reaction of a serum by carrying out the process on a slide with the aid of a rocking movement. Coca demonstrated that cholera vibrio could be agglutinated by means of anti-cholera serum in this way. He described the movements as an imitation of convection currents as they arise in a water bath. Dr. Priestley adapted this technique to the flocculation or precipitation test.[1] He took a slide and divided it into twenty compartments, so that four samples of serum could be tested in four dilutions each with one control for each sample. The antigen employed consisted of an alcoholic extract of human heart and a 1% solution of cholesterin in alcohol in proportions of three to two parts. The dilution of the serum was effected by standardized pipettes and separate pipettes were used for diluting the antigen. The final dilutions of the serum employed in the majority of the tests were 1 in 2, 1 in 5, 1 in 10, 1 in 20 and 1 in 40. In a few instances it was necessary to continue the dilution until 1 in 1,280 was reached. When the serum and antigen mixture in the required dilutions was placed on the slide in the marked divisions, the slide was rotated either by hand or by means of a mechanical rotator for five minutes over an angle of 45° on either side of the horizontal. The results were recorded in three grades. The mere appearance of precipitation visible through a watchmaker's glass was classed as +. When the flocculi were larger, the result was labelled as ++, while +++ was reserved for the largest size of flocculi. The dilution was made with normal saline solution. High concentrations of sodium chloride ranging between 5% and 30% may give rise to spontaneous flocculation. Dr. Priestley applied the test to five hundred samples of serum and compared the results with those of the Wassermann test applied to the same samples. The result of the two tests coincided in three hundred and ninety-five, that is in 79%. The remaining one hundred and five samples are divided into seven groups. In the first group of four samples there was a strong Wassermann reaction and a weak flocculation. The author holds that the former probably reflects the state of affairs more correctly than the latter. In the second group of four samples there was a moderately strong Wassermann reaction, while flocculation occurred twice with 1 in 10 and twice with 1 in 5. He regarded this result as equally favourable for each method. All four patients had been treated some time before. The third group consisted of one sample with a doubtful result to the Wassermann test, but a distinct precipitation in a dilution of 1 in 40 with the flocculation test. As the same serum had previously yielded a reaction to complement fixation, the flocculation was regarded as the more accurate. The fourth group comprised three samples in which the flocculation was more reliable. The fifth group contained fifty-three samples. Dr. Priestley favours the result of the Wassermann test in fifteen and the flocculation in thirty-eight, although he admits some difficulty in arriving at a decision. In the sixth group two samples of sera were tested. Neither reacted to the Wassermann test, but both yielded a reaction in a dilution of 1 in 20 to the flocculation test. Both patients were known to be syphilitic. The last group comprised thirty-eight samples. The Wassermann test either yielded a doubtful response or failed to react, while the response to the flocculation test was very weak in all. The patients had been well treated or had not suffered from syphilis. In summing up, he finds that the diagnosis based on the Wassermann test worked out at 90.8% correct and 9.2% incorrect. When based on the flocculation test by his slide method it was correct in 95% and in correct in 5%.

From information now available it can be stated that the flocculation reaction has a diagnostic significance not less reliable than the Wassermann reaction. The test is easy to perform and it has one great advantage over the Wassermann test, namely the elimination of many reagents. The antigen is not difficult to prepare and it is stable. For the Wassermann test it is necessary to have a fresh suspension of red blood corpuscles, a hæmolytic serum to the same corpuscles, an antigen and lastly fresh complement (guinea pig serum). The notorious unreliability of complement is the chief difficulty in this test. In well equipped laboratories both tests should be applied in cases of doubtful diagnosis. Elsewhere the flocculation test may prove more useful than the Wassermann test.

[1] *The Lancet*, June 23, 1923.

Abstracts from Current Medical Literature.

SURGERY.

Prostatectomy.

H. YOUNG (*Surgery, Gynæcology and Obstetrics*, May, 1923) discusses the pre- and post-operative treatment of prostatectomy based on the study of 1,049 cases treated by the perineal method. The preparation for operation involves preliminary catheter drainage, the use of large quantities of water, tests for renal efficiency by the phenol-sulphone-phthalein method and in certain circumstances blood-urea examinations. Patients in good condition with good phthalein excretion, who have two hundred cubic centimetres or less of residual urine, may be operated on without preliminary drainage. Patients with a larger amount of residual urine, even if the phthalein excretion is good, should have preparatory drainage and forced water treatment, but may often be operated upon in a week if clinical and laboratory studies are propitious. Patients with much impairment of the renal function, 30% or less, should not be operated upon until the phenol-sulphone-phthalein excretion has risen over 40% or until a prolonged treatment for three weeks or more has shown that the optimum has been reached, the condition of the kidneys stable, the blood-urea content not over 0.5 gramme per litre and the general condition fair. Patients with high blood-urea content (over 0.75 gramme per litre) should receive most energetic treatment (seven to ten litres of water by all avenues daily) and the same applies to those with renal infections, especially acute pyelitis. Forced internal hydro-therapy is employed for this condition. Patients with spasmodic, painful or contracted bladder or urethra, some with calculi and some with diverticula, tumour, ulcer or severe cystitis and those in whom catheterization is very difficult or painful or who tolerating a permanent catheter badly, especially if there is suppurative cystitis and epididymitis, often require supra-pubic drainage. In the two-stage operation the first stage is often the more dangerous and is not justified as a routine measure. Patients with high blood pressure may require special pre-operative treatment. With rest in bed, reduced diet and drugs, morphine before operation and ether anæsthesia, the author has found it possible to operate on practically all these patients, even when a history of a previous paralytic stroke has been given. He has also operated on men with severe heart disease, but prefers to use nitrous oxide anæsthesia for these patients. Operation is postponed if any acute inflammation of the respiratory tract is present and when a chronic infection is present gas-oxygen anæsthesia is employed. Some slight improvements in the technique of the author's perineal operation are mentioned. The mortality rate among the 1,049 patients was 3.4%, that is there were thirty-six deaths in all. Of these eight patients died from pneumonia, seven from uræmia, four from pulmonary embolism, three from cerebral hæmorrhage and two from local hæmorrhage. Post-operative shock is guarded against by the administration of an abundance of fluid before operation, by quick operation, careful hæmostasis, packing if necessary, infusion or transfusion of saline solution and, if necessary, blood transfusion. The patient's blood is matched before operation when he is feeble and a donor kept ready. In regard to the deaths from pneumonia a few were from ether pneumonia—which have not occurred since the introduction of nitrous oxide anæsthesia. Pneumonia occurred on several occasions two or three weeks after the operation. This risk is obviated by getting the patients out of bed within a few days and walking within ten days. The risk of uræmia must sometimes be taken; however, by prolonged preliminary treatment and forced water treatment both before and after operation, this risk is minimized. Enemata are not given owing to the risk of pulmonary embolism and the greatest care is taken when there is any indication of phlebitis of the veins of the leg. Epididymitis can usually be controlled by ice packs, but may require incision. Infections of the wound and bladder are treated by mild antiseptic irrigations and infections of the kidneys by internal hydro-therapy. Gauze and drainage tubes are removed from the wound after twenty-four to twenty-six hours. Thereafter the wound is irrigated at each change of drainage. On the fifth or sixth day fluid is forced through the penile portion of the urethra. The passage of sounds is required only when normal urination has not appeared two or three weeks after operation. The author claims that the act of micturition is distinctly more perfect after perineal prostatectomy than after the supra-pubic operation.

Aspiration Treatment of Liver Abscess.

P. MAWSON-BAHR, G. LOW, J. J. PRATT AND A. L. GREGG (*The Lancet*, May 12, 1923) give an account of the aspiration method of treatment of liver abscess. This is not a new method; it was advocated as long ago as 1828. Many authors have described it since that date. The authors give details of its application to fifteen patients. Many of them were acutely ill on admission. There were no deaths, although the mortality is said to be over 50% when treatment by open operation is adopted. The aspiration was combined with the exhibition of emetine-bismuth iodide and ipecacuanha. The authors claim for the method many advantages over open operation. An ordinary Potain's aspirator is used, with a reserve set of tubing in readiness in case the tubing in use becomes blocked. The needle should not be inserted more than 9.5 centimetres. The puncture should be made at the point of greatest tenderness or swelling or in the absence of any localizing signs in the eighth inter-space in the anterior or mid-axillary line. It may also be inserted through the epigastric area. The risk of hæmorrhage is not great, but care should be taken to avoid injury to the lung. As soon as the pus is found, as much of it should be evacuated as possible. If the pus is too thick to flow through the needle, Manson's trocar and cannula should be inserted along the track of the aspiration needle. Injections into the cavity of emetine are not essential. An effusion of serum takes place into the cavity and this is regarded by the authors as the natural method of repair. General anæsthesia is preferable to local, but if the signs are confined to a limited area, local anæsthesia may be used. The authors hold in regard to diagnosis that aspiration should be applied when there is doubt, as abscess may exist without obvious manifestations and in the absence of leucocytosis(pyrexia, the ascertainable presence of *Entamœba histolytica*, pain or tenderness). Pain in the region of the right shoulder may be the most prominent symptom. The diagnosis may be rendered easier by examination with X-rays. Immobility and stretching of the dome of the diaphragm should be regarded as a valuable diagnostic sign.

Mesenteric Thrombosis.

JAMES F. MITCHELL (*Annals of Surgery*, March, 1922) writes about mesenteric thrombosis. He thinks that the condition is better called mesenteric vascular occlusion. The diagnosis is difficult and the results of surgical treatment poor. The occlusion may be venous or arterial, the latter forming 60% of the total number. The veins may undergo a descending thrombosis or the thrombosis may begin in some infective process of the intestine itself or in the appendix. When the artery is involved there may be endocarditis or arteriosclerosis, but in many instances the origin of the embolism is not apparent. The superior mesenteric artery is occluded much more often than the inferior. The affected bowel becomes engorged, later blackened and gangrenous. Blood-stained fluid is found in the peritoneal cavity and also in the lumen of the bowel which becomes distended with gas. The condition if untreated leads to obstruction, perforation of the diseased coil of bowel, peritonitis and death. The symptoms at first resemble those of any intra-abdominal catastrophe. Pain, sudden and agonizing, is the earliest symptom. It is most often situated about the umbilicus or in the epigastrium. The pain is constant and not intermittent as it would be if due to peristalsis. There may be abdominal tenderness and increasing distension. Rigidity is usually absent unless peritonitis has begun. The vomiting is similar to that of intestinal obstruction. Bowel movements are much hindered, but some blood may appear in the stools and this is of great assistance in diagnosis. There is no rise in temperature but a gradual rise

in pulse-rate. In the differential diagnosis the twisted ovarian cyst, volvulus, intussusception, acute pancreatic have to be considered. The treatment is surgical, early operation depends upon early diagnosis which unfortunately is not often made. Resection of the affected bowel leaving a wide margin from the diseased part, with anastomosis of the cut ends, is the most suitable operation.

Glandular Involvement in Cancer of the Caecum.

WINCHELL MCK. CRAIG AND WM. CARPENTER MACCARTY (*Annals of Surgery*, June, 1923) discuss the involvement of the lymphatic glands in cancer of the caecum. In cancer of the caecum dissemination takes places along certain anatomical lines. The lymphatic vessels follow the course of the ileo-colic vessels. The ileo-colic artery is surrounded from its origin to its division by a chain of lymphatic glands. Where the artery branches, this chain is broken into a number of groups situated along the terminal branches of the artery. These groups are classified as (i.) the anterior ileo-colic, (ii.) the posterior ileo-colic, (iii.) the appendicular, (iv.) the ileal and (v.) the right colic. The lymphatic vessels draining the caecum gradually enter one or more of these groups, but occasionally some lymphatic vessels may pass direct to the glands along the ileo-colic artery, an important fact to bear in mind. Cancer of the caecum begins in the glands of the Lieberkühn. Around these glands are numerous lymphatics which form a network and join a second network in the submucosa; the efferent vessels pass to a plexus between the layers of the mesentery. The ileo-caecal valve is involved in about half the cases of cancer of the caecum. The material upon which this paper is based, consisted of one hundred cases of cancer of the caecum treated at the Mayo Clinic. In those in which no involvement of the regional glands took place, the posterior ileo-colic glands were enlarged, in some cases from inflammation, and the same group was attacked most often by cancerous dissemination. Often glands so small as not to be palpable were found to harbour cancerous deposits. In masses of enlarged glands many whose enlargement was due to inflammation, were found sundry cancerous glands, the difference being detected only by the use of the microscope.

Surgical Treatment of Pulmonary Tuberculosis.

EDWARD ARCHIBALD (*Annals of Surgery*, June, 1923) writes of the surgical treatment of unilateral pulmonary tuberculosis. The first landmark in the surgical treatment was the introduction of artificial pneumothorax by Forlanini. In 1909 Freeman, of Denver, advised resection of a few ribs anteriorly over the deceased apex. This was an extra-pleural thoracoplasty, but the operation now indicated by that term is much more extensive. It consists of the resection of the first or second to the tenth or eleventh ribs posteriorly. Various lengths of ribs are removed. The periosteum is left. The operation is always posterior and is begun over the lower lobe and finished over the upper. It may be done in one or two stages. The operation is designed to compress the lung by the inward spring of the ribs and to give the lung physiological rest. The early result is good. Cough and sputum are lessened; appetite and weight increase. Later there may be a relapse and a skiagram will show that some part of lung has not been compressed sufficiently.

Ranula.

VILLARY P. BLAIR (*Annals of Surgery*, June, 1923) discusses the ætiology of ranula. The mucoid cysts which occur about the floor of the mouth, are divisible into two classes. In the first group are included the small cysts which lie in or just under the mucous membrane and protrude partly into the mouth. They arise in various mucous glands in the neighbourhood. The true ranula is placed beneath the mucous membrane and is characterized by a capacity to burrow. It may push the tongue forwards or upwards, but the bulk of the cyst lies in the tissues of the floor of the mouth. It has been stated that such cysts are due to obstruction of the sublingual gland. This would not explain prolongation of the cyst along the lateral wall of the pharynx to the base of the skull. That the cyst is due to a distention of Fleischman's bursa Blair does not believe. He favours the opinion of Thompson that ranula originates in the cervical sinus, that is to say the space produced lateral to the branchial arches by the backward growth of the mandibular and hyoid arches. The growth of the muscles of the neck and floor of the mouth may distort the sinus and pull it in various directions. Consequently the ranula may be bilocular, one part lying under the tip of the tongue superior to the muscles and the other lying in the sub-maxillary region or spreading to the lateral wall of the pharynx. In the latter type removal may be extremely difficult.

Tuberculosis of the Breast.

IN dealing with the subject of tuberculosis of the breast, Dan Collier Elkin (*Annals of Surgery*, June, 1923) remarks on its relative rarity. The condition was described by Sir Astley Cooper in 1829 as "scrofulous swelling of the bosom." Tuberculosis may be primary in the breast or secondary in which case the disease is present in some other part of the body. In primary tuberculosis of the breast the disease would have to be caused by the entry of the tubercle bacillus through the skin of the nipple or through the mucosa of the duct. This is extremely rare. It is most likely that the bacilli arrives by the blood. Some other foci are generally present in the body but overlooked. In the secondary variety the infection may come from axillary glands, ribs, sternum, pleura or lungs. The disease is confined practically to the female, between the ages of twenty and fifty corresponding to the period of reproductive activity. The effect of trauma as a causative factor is not clear. The disease begins as a painless lump in the breast. Pain appears in the latter stages almost constantly. The lump gradually softens, becomes adherent to the skin which assumes a red or purplish colour; then the swelling discharges and a persistent sinus is the result. The axillary glands enlarge; other sinuses appear, so that in the end the breast is discharging from several openings which show no tendency to heal. Usually only one breast is involved. The treatment is removal of the breast and affected glands.

Femoral Hernia.

E. W. HEY GROVES (*The British Journal of Surgery*, April, 1923) gives an account of an operation for femoral hernia. A vertical incision ten centimetres long is made with its centre at the fold of the groin and the sac dissected out. The attachment of Poupart's ligament to the pubic spine is divided and the ligament turned outward, thus exposing the neck of the sac. The sac is cut off and the neck tied. The conjoined tendon is then defined and stitched down to Cooper's ligament and to the upper edge of the pectineal fascia, the femoral sheath on the inner side of the vein being taken up in the outer stitches. Poupart's ligament is then sutured from without inwards to the suture line, the final attachment of the cut end of the ligament lies about 1.25 centimetres further out than originally. Recently, instead of cutting the ligament, a piece of bone has been chipped off the spine of the pubes and at the end of the operation fixed in position with an ivory nail. Twenty-one operations of this nature have been performed by the author and the results have been good. There has been no recurrence.

Teratoid Mixed Tumours of the Breast.

MONROE ANDERSON MCIVER (*Annals of Surgery*, March, 1923) contributes an article on teratoid mixed tumours of the breast. Tumours of the breast containing bone and cartilage have been known for a long time. The growth described by the author contained portion of an alveolar margin with well developed teeth and cartilage. The patient from whom the tumour was removed, was a very obese woman aged sixty years. On account of the hardness of the swelling in the breast a diagnosis of carcimona was made. There is considerable difference of opinion in regard to the ætiology. Wills considers that mixed tumours of the breast are traceable to some abnormality arising at the time of the differentiation of the germ layers. Other authorities think that they arise locally by a process of metaplasia.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Lister Hall, B.M.A. Building, Hindmarsh Square, Adelaide, on July 26, 1923, DR. H. J. RIDDELL, the PRESIDENT, in the chair.

"Insulin."

PROFESSOR T. BRAILSFORD ROBERTSON read a paper by himself and Mr. A. B. Anderson entitled "A Modified Method for the Manufacture of 'Insulin'" (see page 189).

Professor Brailsford Robertson also read a paper by himself and Mr. A. B. Anderson entitled "The Absence of Direct Effect of 'Insulin' Upon the Oxidation of Diacetic Acid in Mice" (see page 191).

DR. C. T. CH. DE CRESPIGNY read a paper entitled "The Treatment of Diabetes with 'Insulin'" (see page 193).

The discussion on these papers was postponed until the next meeting. Dr. H. J. Riddell said that it had been proposed to address letters to the Manager of the Metropolitan Abattoirs and to the veterinary surgeon as well as to the Secretary of the Abattoirs Board conveying the thanks of the South Australian Branch of the British Medical Association for their help in providing the material for the production of "Insulin." Without the assistance of these gentlemen this important work could not have been carried out. The proposal was adopted.

THE PRESIDENT thanked Professor Brailsford Robertson for his most interesting papers.

The Pancreas in Diabetes.

PROFESSOR J. B. CLELAND exhibited several specimens of pancreas of persons who had died of diabetes. The first was from a patient of Dr. de Crespigny. She had died of diabetic coma unaffected by "Insulin." There was no obvious naked eye changes in the organ. Microscopically no definite changes had been found in the islands of Langerhans. The islands themselves were more numerous towards the tail than towards the head. The nuclei of the cells were well preserved and spherical. The tubules of the kidneys and the Malpighian corpuscles were dilated.

The second specimen was from a patient of Dr. W. Ray. The patient had died at the age of thirty-two years from diabetic coma unaffected by "Insulin." The pancreas seemed a little smaller than normal. Tracheitis, bronchitis, some broncho-pneumonia and pus in both middle ears and in the ethmoidal cells were detected. In the microscopical sections of the pancreas well preserved islands of Langerhans were seen, perhaps fewer than normal, but well supplied with capillaries. There was no obvious change in the cells. The vessels to the pancreas were rather thick walled.

The third specimen was from a child, aged five years and a half, under the care of Dr. de Crespigny at the Children's Hospital, in July, 1920. Death had taken place from diabetes. No obvious change had been detected in the organ. The cells in the islands were arranged in an indefinite but easily recognizable tubular fashion. This indicated a failure of the islands to mature properly and this failure was accompanied by physiological insufficiency.

The fourth specimen was from a woman, aged sixty-seven years. She had been maniacal and then comatose before death. She had been under the care of Dr. Swift in the Adelaide Hospital in 1911. The pancreas was described in Dr. de Crespigny's notes at the autopsy as being much wasted, tough and fibrous. The specimen looked as though there had been much fatty infiltration.

Acute Hæmorrhagic Pancreatitis.

Professor J. B. Cleland also showed a specimen of a pancreas from a patient, aged sixty-three years, who had been under the care of Dr. de Crespigny. The woman had died from heart disease and the existence of the acute hæmorrhagic pancreatitis had been discovered at the autopsy. She had complained of severe abdominal pain before death. No blockage had been detected at the ampulla of Vater. The kidneys had presented a pallid, dull greenish, glassy aspect on section. Microscopical examination had revealed a clear material filling the lumina of many of the tubules and continuous with the peripheral parts of the cells, many of whose nuclei were still well preserved in the basal parts. Other tubules were filled with polymorpho-nuclear leucocytes indicating an infection. The relation, if any, between the kidney lesions and the pancreatic lesions was not clear.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Brisbane General Hospital on July 6, 1923, DR. EUSTACE RUSSELL, the President of the Brisbane General Hospital Clinical Society, in the chair.

Odontome.

DR. E. CULPIN showed a man, aged twenty-seven, suffering from an odontome of the upper jaw. In 1909 he had had an abscess of the upper jaw and a tooth had been extracted. Early in 1923 another molar had been extracted and a persistent sinus had resulted. A skiagram revealed an odontome in the upper jaw; the antrum was clear.

Peripheral Neuritis Following Acute Rheumatism.

DR. EUSTACE RUSSELL presented a girl, aged sixteen, with a history of sudden paralysis of the arms. On examination the condition had been found to be due to generalized acute articular rheumatism. There had been no paralysis. Under salicylate treatment all the joints had recovered, except one ankle joint. There was a considerable neurotic factor in the case. When the girl was allowed out of bed there was a noticeable foot drop. That there were joint lesions had been demonstrated by X-ray examination.

DR. S. F. MCDONALD considered the foot drop was largely functional. There was a curious inturning of the foot not seen in ordinary foot drop. The foot was very tender and there was no anæsthesia. The electrical reactions had not been mentioned.

DR. T. H. R. MATHEWSON stated that the condition of the foot had apparently only been noticed after the patient had got out of bed. It was not a case of ordinary foot drop; the foot was held too rigidly. He considered it was largely a functional condition.

DR. RUSSELL in replying stated that at first he had considered the case was purely functional and had treated it as such, but the patient complained of so much pain that it prevented him insisting on her walking earlier. He did not desire to force the girl to walk, if this caused much pain.

Bilateral Polycystic Kidneys.

DR. A. H. MARKS presented the kidneys and liver of a patient who had polycystic kidneys. The patient, a married woman, a nullipara, aged thirty-four, had been admitted with a history of having fainted. She had been semi-conscious; the pupils had been unequal and the breathing had been laboured. The abdomen had been filled with three tumours, in both loins and in the middle line below the umbilicus. The urine had a specific gravity of 1,0005 and contained a small amount of albumin. The cerebro-spinal fluid had been clear and under slightly increased pressure. There had been œdema of the optic discs. She had rapidly become worse and had died. At the *post mortem* examination the transverse tumour was found to be the lower pole of the left kidney. The liver contained a few cysts also.

Vesical Calculus.

Dr. A. H. Marks showed a large calculus removed from an elderly woman who had been suffering from *procidentia* and prolapse of the rectum.

Epithelioma of the Face.

DR. LOCKHART SPENCE, M.B.E., showed two patients to illustrate the results of diathermy for epithelioma of the face. In one a lymphocele had developed in the scar.

DR. V. MCDOWALL stated that many of their patients had been sent to the Hospital in an inoperable condition and had been treated as a last hope. Diathermy was the only method which gave relief and in some cases this was permanent after X-rays, surgery and radium had failed. The relief from pain was great.

DR. D. A. CAMERON asked to what depth diathermy could be applied in order to remove the outer plate of bone and still leave the inner intact.

DR. SPENCE in reply stated that as regards the soft structure, this was controlled by the size of the electrode and the spark gap. There was no gauge for removing the outer plate of bone alone.

Band Causing Obstruction of the Duodenum.

DR. A. G. ANDERSON showed a man, aged fifty-eight, who had suffered from pain in the epigastrium radiating backwards between the shoulders. The pain had occurred after food and had occasionally been continuous. He had been very constipated. Nothing definite had been discovered on examination. A radiogram showed dilatation of the duodenum due to some obstructive cause. At the operation a band the thickness of a finger had been found stretching from the tip of the gall bladder across the descending portion of the duodenum to the omentum above the transverse colon. There had been no attachment directly to the duodenum. The band had been severed and all symptoms had disappeared. Radiograms taken after the operation showed the stomach in normal position and the duodenum small and not enlarged.

Persistent Bone Sinuses Following Acute Osteo-myelitis.

Dr. A. G. Anderson presented a boy, aged twelve, who had been admitted two years previously to the Hospital for Sick Children with osteo-myelitis of the upper end of the tibia. The knee joint had been perforated and the joint full of pus. There had been much infection and numerous operations for removal of sequestra. The patella had been excised. He had thoroughly excised the persistent sinuses and as the overlying soft tissues were insufficient to fill the gaps, had utilized flaps from ths soleus muscle. The result promised to be very successful.

Syphilitic Ulceration Throat.

DR. C. E. TUCKER showed a patient suffering from ulceration of the throat. The patient had been complaining for six months of an ulcerated condition of the left lower gum, stated to be due to a scratch from a tooth brush. There was a left-sided peritonsillitis and a small opening in the tonsil exuding pus. The ulcerative condition extended from the lower left canine to the last molar and was covered by an unhealthy slough; the edges were thickened and indurated. No glands were palpable. The ulcer had been curetted and cauterized with pure carbolic acid. The condition was slowly spreading and numerous small glands had become palpable on both side of the neck. There was a history of gonorrhœa five years previously. No Wassermann test had been applied.

The general opinion of the members was that the lesion was syphilitic.

Tumour of the Sternum.

DR. E. S. MEYERS presented a man, aged seventy-three, with a tumour of the sternum. Previously a prostatectomy had been performed. The swelling, situated between the second rib and sternum, had been hard and egg-shaped in outline. The heart was normal. The patient's serum did not yield a reaction to the Wassermann test. Administration of potassium iodide had made no difference. A diagnosis of chondroma had been made and the patient had been discharged. On readmission there had been a diffuse tumour with a sinus leading down to dead bone. A radiogram showed a large area of rarefaction of the first rib and sternum. The condition was either a secondary focus from the prostate, tuberculosis, syphilis or sarcoma. He intended to explore the swelling.

MEDICO-POLITICAL.

THE COUNCIL OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION passed the following resolution at its meeting on August 15, 1923:

> No alteration or addition to the treatment of a patient already under the care of a medical man shall be made by another medical man acting in his capacity for an insurance or other company or by any other officer of that company without first consulting or discussing the matter with the medical man in charge of the case.

This resolution has reference to persons in hospital or other institution as well as those under the private care of a medical practitioner.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

BURSTAL, ALBERTO CHARLES CLUBBE, M.B., Ch.M., 1921 (Univ. Sydney), Coast Hospital, Little Bay.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

BROWN, HAROLD GILLBEE, M.B. *et* Ch.B., 1917 (Univ. Melbourne), 230, North Road, North Brighton.
HIRSCHFELD, OTTO SADDLER, M.B., B.S., 1923 (Univ. Melbourne), Melbourne Hospital.
REID, COLIN CAMPBELL, M.B., B.S., 1923 (Univ. Melbourne), Melbourne Hospital.
WATERHOUSE, GEORGE ALAN, M.B., B.S., 1923 (Univ. Melbourne), Melbourne Hospital.
WOODS, JOHN GRIEVE, M.B., B.S., 1923 (Univ. Melbourne), Alfred Hospital, Prahran.

Congress Notes.

THE PAN-PACIFIC SCIENCE CONGRESS.

WE are informed that two alterations have been made in the programme of the Sydney meeting of the Section of Hygiene of the Pan-Pacific Science Congress.

In the first place the general discussion on "The Organization and Coordination of Scientific Work in the Pacific Region" will be held on Tuesday, August 28, 1923, instead of Friday, August 31, 1923. On the latter date there will be a discussion on "Settlement in Areas of Low Rainfall."

Professor H. E. Gregory will open the discussion on the organization of scientific work from the point of view of geography. The subsequent speakers will be Dr. Guy A. K. Marshall (Entomology), Professor E. C. Stakman (Agriculture), Dr. J. H. L. Cumpston (Hygiene), Dr. E. D. Merrill (Botany), Dr. A. C. Haddon (Anthropology), Colonel Sir Gerald Lenox-Conyngham (Geodesy), Dr. Van Braak (Geophysics), Dr. T. Wayland Vaughan (Geology), Professor C. M. Fraser (Zoology) and Dr. S. Dodd (Veterinary Science).

The discussion on the settlement with low rainfalls will be opened by Professor Ellsworth Huntington. No representative of the Section of Hygiene will take part.

Obituary.

GEORGE EDWARD RENNIE.

BUT a few weeks ago the Queensland Branch of the British Medical Association made preparations to receive Dr. George Edward Rennie at a meeting of the Branch on July 5, 1923, at which he was to have given an address on glycosuria and diabetes. The Council had further invited him and Dr. W. H. Crago to be the chief guests at the annual dinner and one of the members had been chosen to give the toast of the evening, "Our Guests, coupled with the name of Dr. George E. Rennie." A few

days before the date, the "chief guest" was taken ill and the engagement had to be cancelled. On August 10, 1923, this brilliant neurologist died of a neurological affection, a thrombosis of a cerebral vessel. Far beyond the confines of New South Wales the news of his death has caused feelings of the deepest regret. His contemporaries mourn because they have lost a colleague whom they revered and admired and a friend whom they trusted; his erst-while students recognize that his death means something more than the passing of a man who at one time taught them part of what they had to learn. They owe much to his influence, precept and guidance, as well as to his excellent teaching and they feel that the vacant place of counsellor, judge and friend cannot be filled. At the grave-side when he was laid to rest there was a great congregation of grieving men and women, an eloquent testimony of the value each and everyone placed on his life and his life's work.

George Edward Rennie was born on September 10, 1861, in Sydney. His father was Auditor-General of New South Wales. He was educated at the Sydney Grammar School. From an early age he revealed an unusual aptitude in the class room and won the golden opinions of his teachers. He matriculated at the age of eighteen and passed into the University of Sydney where he gained prize after prize. In 1897 he obtained the Salting Exhibition and a general scholarship. In 1880 he won the Levy Scholarship and in the following year the Deas-Thomson and the Want Scholarships. Two years later he gained the Belmore Gold Medal and the Gilchrist Scholarship. He took his degree in Arts in 1882 with first-class honours in Greek and Latin and in natural science. In the same year, he went to London and entered the University College Medical School. Here again he distinguished himself by his brilliant intellectual qualities and his versatility. He won prizes in anatomy, chemistry, physiology, *materia medica*, medicine, surgery, ophthalmology. The Bruce Gold Medal and the Aitchison Scholarship fell to him during his student days. In his examinations he was equally successful. He gained first-class honours in zoology in the preliminary M.B. examination. His career was unchecked throughout the whole course. In 1887 he took his degree of Bachelor of Medicine and in the same year he passed the examination for the diploma of member of the Royal College of Surgeons of England. In 1888 he obtained his Doctor of Medicine with the high distinction of the Gold Medal. It is said that he was the first Australian to gain this coveted prize in London.

At a later date he returned to London and passed the examination of the Membership of the Royal College of Physicians of London, a searching and at that time (1899) infrequently taken examination. Eight years later the Council and Fellows of the College elected him a Fellow, a distinction zealously guarded and distributed only to those whose scientific attainments in medicine are of a high order. There were then but very few practitioners in Australia who had gained this honour and even today their number is very small.

George Edward Rennie returned to New South Wales in 1889 and immediately mapped out for himself a career for which he was preeminently fitted. He held a position at the Sydney Hospital for a time and then began his long association with the Royal Prince Alfred Hospital. First he was appointed Assistant Pathologist. In 1894 he was elected an Honorary Assistant Physician. In his activities in this capacity he turned his attention to neurology and psychiatry and soon he established for himself a reputation as one of the leading authorities in diseases of the nervous system in his State, perhaps in Australia. As time went on, this reputation gained in weight and for many years his position as a leader of thought in this branch of study has been unchallenged. In 1898 he was promoted to the position of Honorary Physician. At this time his private practice was considerable and he had already made many important contributions to medical literature. The majority of these contributions were on nervous diseases, such as Friedreich's ataxy, muscular hypotonia and so on. As indicated above, George Edward Rennie paid a visit to England in 1898, when he sat for the examination for the membership of the Royal College of Physicians. On his return to Sydney in 1900, he again joined the medical staff of the Royal Prince Alfred Hospital, but for six years acted again as Honorary Assistant Physician. In 1906, he became full Honorary Physician. Since 1912 he was Senior Physician until his retirement at the age of sixty years, when he was placed on the consulting staff.

In 1898 an institution was opened in close proximity to the Reception House, at Darlinghurst, for the treatment of persons suffering from mental affections with good prospects of rapid recovery. The Mental Hospital differed from the hospitals for the insane in that the patients were not subjected to certification and therefore were not retained in the institution against their wishes. During the fourteen years of its existence, George Edward Rennie performed valuable work in this institution as a Visiting Physician. The work was done, like all his work, without ostentation and without any thought of gain to himself.

George Edward Rennie was for some time Lecturer in Medicine at the University of Sydney and for many years Lecturer in Clinical Medicine at the Royal Prince Alfred Hospital. As a teacher he excelled because he possessed knowledge and knew how to impart it. His method of bed-side teaching was unequalled and his students soon learned that the serious, almost stern and unyielding manner of compelling them to think instead of repeating in parrot-fashion the text-book phrases was a kinder way than any other. He had a wonderful power of diagnosing obscure and atypical conditions and his grasp of morbid anatomy was unusually wide and far-sighted. He combined the qualities of physician with those of physiologist and thus justified his position as a medical scientist. Little wonder that his students have always proclaimed the fact with pride that they derived their training at the hands of this great master.

Early in his medical career, George Edward Rennie took an active part in the work of the New South Wales Branch of the British Medical Association. He joined shortly after he started in practice. In 1896 he was elected by his colleagues to the Council of the Branch and his re-

election followed with clock-like regularity for eighteen years. From 1897 to 1906 he acted at Honorary Secretary of the Branch. He became Vice-President in 1901 and President in 1902. In each office he worked strenuously and to the great advantage of the organization and earned well merited appreciation and praise.

In January, 1901, Dr. Samuel Knaggs relinquished the position of editor of the *Australasian Medical Gazette*, the organ of the New South Wales Branch of the British Medical Association. The following nine monthly issues were produced by Dr. W. H. Crago, who undertook to occupy the position of acting editor pending a permanent appointment. In October the rules of the Branch were amended to enable the Council to make the appointment and George Edward Rennie was immediately selected for the post. While he continued to practise both in hospital and in private, he edited the *Australasian Medical Gazette* for over twelve and a half years. For ten years this publication appeared once a month, but in 1912 it became a weekly journal. It is impossible to speak too highly of the manner in which the work was carried out. His general scholarship, his sound knowledge of clinical medicine, neurology and pathology and his extraordinary ability to work at high pressure enabled him to raise the standard of excellence of the *Gazette* and to maintain that excellence for a long series of years. At the same time, he contributed many signed articles on the subjects that he had made specially his own, not only in the *Gazette*, but also in other medical journals. In 1914 when the Federal Committee of the British Medical Association in Australia had founded the Australasian Medical Publishing Company, Limited, so that the medical profession in Australia might have a federal journal, the Council of the New South Wales Branch recorded its indebtedness to George Edward Rennie at a dinner and in two annual reports. The volumes of the *Australasian Medical Gazette* stand as a monument of his industry, ability and efficiency.

There is another picture to be drawn of the character of George Edward Rennie. In his family circle and to his intimate friends he was the embodiment of healthy manhood, kind and courteous, bright and homely. His attachments were very deep and lasting. As an ideal husband and father, he brought happiness into his home and found it there. He was a man of high ideals, religious and intensely honest. He could fight hard, but he fought fairly. In debate, in the columns of the *Gazette*, in his whole life he maintained his principles in spite of the most ingenious and provoking argument or opposition. He hated sarcasm and satire was foreign to his thought.

Two of his sons served with distinction in the Great War and both made the supreme sacrifice. Cyril Rennie obtained a commission as Lieutenant in the Leicestershire Regiment and was killed in France in the year 1916. Clement Rennie also secured a commission. He joined the Royal Air Force and was killed at Salonica within a few months of the end of the war. The death of his two boys was an almost intolerant blow to George Edward Rennie and impressed upon him an indelible mark. He is survived by his widow, two sons and two daughters, to whom the sympathy of the whole medical profession is extended.

DR. W. H. CRAGO writes:

For over thirty years Dr. Rennie and I have enjoyed the closest personal friendship. We have been intimately associated in practice, we were colleagues on the Council of the New South Wales Branch of the British Medical Association for sixteen or seventeen years, for thirteen years we were associated as Editor and Manager respectively of the *Australasian Medical Gazette*, for many years we have been fellow members of the Medical Board of New South Wales and we have been travelling companions on many short trips to Hobart and Melbourne and other interstate ports, so I can unhesitatingly state that his death will cause a great blank in my life.

Dr. Rennie was a man of deep religious convictions. He was a devout believer in the Holy Scriptures and maintained throughout his life the practice of family and public worship. As a medical practitioner and consultant he was thorough in all his work, sparing no pains to investigate carefully the condition of every patient that came under his care. His greatest labour of love was undoubtedly his work at the Royal Prince Alfred Hospital, extending over many years. He was scrupulously regular in his attendance and with what success he performed his duties can be testified to by patients that have been under his care and by many medical practitioners throughout Australia who have been his pupils. It was a great grief to him when the age limit necessitated his retirement. He highly appreciated the opportunity that was afforded to him of ministering to some of the returned soldiers at the Randwick Military Hospital. Dr. Rennie's manner was naturally reserved and unassuming, but when he admitted anyone to his friendship, all reserve disappeared and he became a most congenial companion. The loss of two of his sons in the war was a source of profound grief to him, from which he never completely recovered. As a friend he was ever ready to give good counsel and was unwavering in his steadfastness. His scholastic attainments, as recorded in the *Sydney Morning Herald*, of August 11, were phenomenal and he remained a student to the last. He had promised to read a paper on diabetes before the Queensland Branch of the British Medical Association on July 6, on which trip I was to have accompanied him, but unfortunately his illness frustrated his plans. I conclude with the words of the Psalmist: "We took sweet counsel together and walked in the house of God as friends."

DR. H. J. CLAYTON writes:

In the grave loss which the State has sustained through the death on August 10, 1923, of one of its most eminent, though most retiring and unselfish citizens, the medical profession and the Royal Prince Alfred Hospital most poignantly share.

To the junior members of the medical profession, whose rôle it is to follow the teachings of their masters, Dr. Rennie was an inspiration to excel, a brilliant intellect, a devoted student of his art, a methodical clinician and an intensely conscientious and loyal servant to his patients and to the medical services with which he was associated.

Of the many to whom he was known and by whom his professional attainments were admired, not a great number were privileged to know him closely, for his was not the personality which claimed acquaintances as friends nor sought a bubble reputation through social avenues. I think I can, however, count myself one of those fortunates among the junior members of the profession, who knew Dr. Rennie intimately from long association with him in various capacities and, because my admiration for his mentality and the quality of his work made this association one always to be sought, I came to know and to admire the more.

My first introduction to his methods was as a medical student in 1910, when Dr. Rennie was acting as Lecturer in Medicine at the Sydney University. He was undoubtedly the best systematic lecturer I have ever listened to, his lectures showing in their delivery the hours spent on their preparation and a unique power of coordination and systematic presentation, evidence of the master mind, organized and thorough.

On graduation I became Dr. Rennie's House Physician at the Royal Prince Alfred Hospital and here I learnt the genius of the master, the thoroughness of his clinical methods and his acumen in diagnosis, particularly of conditions seen rarely even in a large public hospital, yet with every detail of which he was conversant and which he delighted to investigate and to discourse upon.

Of the simplest case this extraordinary mind could always present some unconsidered feature, providing always mental pabulum where the average man would remark nothing of interest and so, as House Physician, Senior Resident and as Medical Superintendent of the Hospital over a period of many years and more recently, too, as Honorary Medical Officer, I was wont to obtain stimulus by learning his views on clinical material and where possible deferring difficult or anomalous cases to him.

His clinical and scientific excellence was enhanced by his profound and minute knowledge of morbid anatomy and every disease seen in his clinic conveyed to him a

mental picture of the pathological condition present—a picture which he portrayed to his students. He was invariably present at autopsies on any of his cases which died, and his correlation of clinical facts to pathological findings conveyed more instruction than a mutitude of theories. Would that more of our teachers were following in his footsteps!

His was the personality which by its own thoroughness, exactness and devotion to the work in hand, inspired similar qualities in those with whom he laboured, and I found always that the most conscientious loyal medical officers were those who were associated with Dr. Rennie. He demanded nothing, but he received the type of service he himself gave. And so it was, too, with the nursing staff whom he inspired with his own zeal and interest. He trusted those who were working for him, and received full measure.

No demand was too great for Dr. Rennie in his services to the institutions with which he was connected. He was the ideal honorary medical officer, not only in the direction of sustaining, but of expanding the activities of the Hospital. Apart from his routine services, he devoted his private time in innumerable ways and the hours of his private practice were always available if called on.

During the influenza epidemic, when the Hospital passed through the greatest medical crisis of its history, he threw himself with all his skill and energy into its efforts; and not only did a large share in the treatment and investigation of the epidemic, but supervised the treatment of the casualties among the medical and nursing staff, numbering at one time over a hundred. In those weeks he attended for hours daily—sometimes on two or three occasions in one day for those who were dangerously ill—and his attention was unflagging and unremitting.

His career was one of many disappointments. He was no politician; he knew no subtleties or bypaths; and had he been less earnest and less devoted to his object of doing the thing because it was worth doing, he might have secured more advancement in material directions. But I am sure that he received his reward in the knowledge of work well done and in the knowledge that his brilliant intellect and unswerving devotion to his duty excited admiration in and were an example to his associates. His epitaph should truly read: "Well done, thou good and faithful servant."

One thing we can do to remember and to honour this great citizen and member of our profession. It is to our discredit—nay, to our dishonour—that the medical institutions with which our great men have been associated and to which they have given their best services throughout the whole of their lives, have neglected the obvious duty of endeavouring to repay their debt by making reasonable efforts to advance the careers of the sons of these men. Surely it can be claimed that the medical sons of such men are entitled to every opportunity of securing such benefits as these institutions can give them—primarily at least, by appointment to the resident medical staff and the opportunity then of advancement to more senior positions. What greater or more simple mark of respect or of remembrance can be paid? It cannot be argued that such a step "will create precedent." The number of cases in which it need be done, is so small as to be but a slight departure and, indeed, such precedent must always be a good one to follow. There are so few truly great men that surely we cannot create many precedents!

Dr. Rennie was a man of God and that he had done well was to him sufficient reward. But we, who are less worthy but perhaps more worldly, should see to it that such a labourer should get his posthumous reward in the advancement of the son's career in the profession which his father adorned.

WILLIAM REATH OLVER.

IT is with regret that we announce the death of Dr. William Reath Olver which took place at Chatswood, New South Wales, on August 15, 1923, at the age of seventy-four.

Correspondence.

THE VISIT OF SIR WILLIAM MACEWEN.

SIR: In accordance with a recommendation of the Executive Committee of Congress, I beg you to publish this letter in your next issue.

Will all former students, house-surgeons or assistants of Sir William Macewen please communicate with the undersigned?

It is suggested that all such might wish to combine in a united welcome to the old "Chief"? Suggestions will be appreciated.

Yours, etc.,

C. DUGUID.

192, North Terrace, Adelaide,
August 13, 1923.

THE HICKSON MISSION.

SIR: The Healing Mission Committee appointed in connexion with Mr. Hickson's late visit to Adelaide, are anxious to enlist the assistance of medical men, who are in a position to supply accurate data of cases treated at that mission. Any records, with reports on the subsequent progress, will be highly appreciated and may be sent to the undersigned.

Yours, etc.,

J. W. YEATMAN.

Old Beach Road,
Brighton, South Australia,
July 30, 1923.

THE RESULTS OF TONSILLECTOMY.

SIR: In the edition of THE MEDICAL JOURNAL OF AUSTRALIA of August 4, 1923, some statistics on the results of tonsillectomy by Dr. Walter C. Alvarez were quoted. At first sight not too favourable deductions might be drawn from these statistics as to the value of tonsillectomy, but closer observation may expose their fallacies. He quotes that of seventy-six persons operated on for recurrent tonsillitis fifty-seven claimed that the result was good. The remainder were all definitely improved. If their tonsils had been enuncleated completely, it was quite impossible for them to have had any recurrence.

Of seventy-two persons with tonsillitis and sore throat twenty-three claimed a good result, thirty-three were definitely improved and sixteen were not improved. Or this series it must be quite obvious that the operator could not have completely enucleated the tonsils, such being the case very little improvement could be expected. Even where enucleation has been successfully carried out, there still may remain infection in the lingual tonsils; also there are small patches of lymphoid tissue scattered over the pharynx which are capable of carrying on the infection.

Of one hundred people who had no tonsillitis, but were operated on for other reasons, three claimed a good result, sixteen showed some improvement and the remainder received no benefit. It is quite a remarkable thing that some men who hear that rheumatism can arise from such conditions as tonsils, when a patient goes to them with rheumatism, say: "You had better have your tonsils out," regardless of the fact that the infection was probably coming from pyorrhœa and *vice versa*. This sort of thing is too painfully common at the present time. That individual then goes about saying: "All this question of tonsils is much overdone."

To continue: It is interesting to note that of forty-seven persons suffering from "rheumatism" seven claimed a good result, five obtained relief, thirty-one obtained no relief and the remainder were only relieved temporarily or became worse. There is a kind of assumption here that infected tonsils were the only source of rheumatism. For those that have had some experience in these matters,

it is quite obvious that pyorrhœa is the dominating element in adults in the production of arthritis, whereas tonsils are in children. If a person has pyorrhœa or a suppurating antrum, can any benefit be expected from removal of his tonsils? A very large proportion of patients with infected tonsils have infected sinuses as well, the same organism invades both and unless the sinus condition is cleared up, no benefit will result from the removal of the tonsils. It is not a matter of simply removing tonsils; it is a matter of cleaning up breeding places of bacteria, wherever they may be.

There is nothing to prove that the tonsils are in any way beneficial to us, by stimulating our defence mechanism. It is a popular idea which seems to have hypnotized the general mind. People with tonsillar infection are not healthier than those without. They would be, if this premiss was true. It is far more likely that the phagocytic action which goes on within the tonsils, is primarily meant as a destructive action to the bacteria gaining access, but that in time from chronic infection, bacteria have taken to multiply within the crypts and the white cells have failed to completely protect the host, enabling them to gain access to the blood.

Before statistics of this nature are placed before the medical world from which to draw conclusions, great care must be taken in mentioning all the infected points in the case, as it will be found that in many cases they are multiple.

The question is: Is it wise to breed virulent bacteria in one's tonsils or not? Until this question is answered to the satisfaction of everybody, we can get no further with the tonsil problem. At present every man has a different view of what is an infected tonsil, very few do the only obvious thing and take a culture to see what is in the prize-packet. On the finding of virulent bacteria in the tonsillar crypts (and we know they mostly contain them), the man is going to take a big responsibility upon his shoulders who can advise his patient to let them remain there.

Yours, etc.,

SYDNEY PERN.

Melbourne,
August 13, 1923.

INSTRUCTIONS GIVEN TO SYPHILITIC PATIENTS.

DR. W. MCMURRAY, of 175, Macquarie Street, Sydney, has sent us the following translation of the letter-press on a card of instructions given to patients attending the clinic for syphilis at the Charité Hospital in Berlin. It is an interesting document.

> Your disease is contagious; you must, therefore, be careful not to transmit it to others by kissing or by other close contact, by sleeping with others in the same bed or by using at the same time with others the same eating and drinking vessels. Your disease is not cured by a single course of treatment. You will probably at some time again discover something of your disease (for instance, open sores or pain in the mouth or throat or in the sexual organs or eruption in the body). As soon as you notice any such appearances, you must at once again undergo medical treatment, but also when there is nothing noticeable, you should see a doctor about once a month. The treatment used need not necessarily take place at the hospital, as you will probably be able to continue your work. After about three years of proper medical treatment applied several times a year, you will then very likely be spared any later disease symptoms, for instance, caries, early apoplexy of the brain. Years after infection and only by permission of your medical adviser you may marry, otherwise you might transmit the disease to your wife (husband) and to your children.
>
> Your disease is certainly curable by proper medical treatment. Keep this card by you and show it to your doctor whenever or for whatever illness you may have to consult him, but do not show it to anybody else.

Naval and Military.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Nos. 47, 52, 54 and 56, of July 5 and 26 and August 2 and 16, 1923.

Australian Military Forces.

SECOND MILITARY DISTRICT.

Australian Army Medical Corps.

MAJOR C. L. CHAPMAN, D.S.O., is restored to the authorized establishment of Majors, 1st November, 1922.

THIRD MILITARY DISTRICT.

Australian Army Medical Corps Reserve.

HONORARY CAPTAINS H. F. LAWRENCE AND C. H. MOLLISON are retired under the provisions of Australian Military Regulation 152 (1), 4th August, 1923.

FIFTH MILITARY DISTRICT.

Australian Army Medical Corps Reserve.

HONORARY CAPTAINS C. W. LAVER AND F. L. P. SAWELL are retired under the provisions of Australian Military Regulation 152 (1), 17th June, 1923, and 28th June, 1923, respectively.

Retired List.

The names of MAJORS J. B. LEWIS, K. R. DE V. SHAW AND W. S. SWEET, CAPTAINS G. S. LANDON, H. O. CHAPMAN, A. C. OLIVER AND A. J. MCSHANE are removed from the Retired List under the provisions of Australian Military Regulation 159, 17th May, 1923.

Australian Army Medical Corps.

To be Captain (provisionally).—ADRIAN WARD FARMER, 1st August, 1923. The provisional appointment of CAPTAIN A. W. FARMER is terminated under the provisions of section 15 of the Defence Act, 31st July, 1923.

CAPTAIN R. H. CRISP is appointed from the Reserve of Officers, 1st July, 1923; CAPTAIN J. KENNY is transferred to the Reserve of Officers and to be Major, 30th June, 1923.

SIXTH MILITARY DISTRICT.

Australian Army Medical Corps Reserve.

HONORARY CAPTAIN J. L. KERR is retired under the provisions of Australian Military Regulation 159, 21st May, 1923.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned have been registered under the provisions of the *Medical Act 1915*, as duly qualified medical practitioners:

KERR, JOSHUA LAW, M.B. *et* C.M., 1880; M.D., 1890 (Aberdeen), 29, Blyth Street, Brunswick.

THOM (MRS.), EDITH MARJORIE, M.R.C.S. (Eng.), L.R.C.P. (Lond.), 1918. "Eildon," Grey Street, St. Kilda.

Additional Qualifications Registered.

GREEN, JOHN SYDNEY, M.D. (Melb.), 1922; Dip. G.O. (Dublin), 1920.

Names of Deceased Practitioners Removed from the Register.

ACTON, FREDERICK CHARLES.
AITKEN, WILLIAM LEWIS.
KENNEDY, PATRICK.

Books Received.

A TEXT-BOOK OF DENTAL ANATOMY AND PHYSIOLOGY, by John Humphreys, M.D.S. (Birm.), F.S.A., F.G.S., F.L.S. and A. W. Wellings, M.D.S. (Birm.), L.D.S. (Edin.); 1923. London: Edward Arnold and Company; Demy 8vo., pp. viii. + 323, with 254 illustrations. Price: 16s. net.

A PRACTICAL HAND-BOOK OF DISEASES OF THE EAR (FOR STUDENTS), by William Milligan, M.D. and Wyatt Wingrave, M.D.; 1923. London: William Heinemann (Medical Books), Limited; Demy 8vo., pp. 191, with 72 illustrations.

THE COMMON NEUROSES: THEIR TREATMENT BY PSYCHOTHERAPY, by T. A. Ross, M.D., F.R.C.P.E.; 1923. London: Edward Arnold and Company; Demy 8vo., pp. xi. + 256. Price: 12s. 6d. net.

THE NATURAL PHILOSOPHY OF LOVE, by Remy de Gourmont, Translated with a Postscript by Ezra Pound; 1922. New York: Boni and Liveright; Sydney: Angus and Robertson, Limited; Demy 8vo., pp. ix. + 222. Price: 25s. net.

MEMOIRS: WITH A FULL ACCOUNT OF THE GREAT MALARIA PROBLEM AND ITS SOLUTION, by Ronald Ross; 1923. London: John Murray; Sydney: Angus and Robertson, Limited; Demy 8vo., pp. xi. + 547, with eleven plates. Price: 24s. net.

TUTANKHAMEN: AND THE DISCOVERY OF HIS TOMB BY THE LATE EARL OF CARNARVON AND MR. HOWARD CARTER, by G. Elliot Smith, M.A., M.D., Litt.D., F.R.S.; 1923. London: George Routledge and Sons, Limited; Sydney: Angus and Robertson, Limited; Crown 8vo., pp. 133, with twenty-two illustrations. Price: 4s. 6d. net.

THE HYGIENE OF MARRIAGE, by Isabel Emslie Hutton, M.D.; Foreword by Professor A. Louise McIlroy, M.D., D.Sc., O.B.E.; 1923. London: William Heinemann (Medical Books), Limited; Crown 8vo., pp. xii. + 112. Price: 6s. net.

CANCER: FALLACY, THEORY AND FACT, by John Shaw, M.D. (Lond.); 1923. London: George Routledge and Sons, Limited; Sydney: Angus and Robertson, Limited; Demy 8vo., pp. 191, with statistical diagrams, tables and illustrations. Price: 12s. 6d. net.

ENCYCLOPÆDIA MEDICA, under the General Editorship of the late J. W. Ballantyne, M.D., C.M., F.R.C.P.E. (Volumes I.—VIII.) and Alexander Goodall, M.D., F.R.C.P.E. (Volume IX.); Second Edition; Volume IX., Mammillaplasty to Obesity; 1923. Edinburgh and London: W. Green and Son, Limited; Sydney: Butterworth and Company (Australia), Limited; Royal 8vo., pp. 672.

SURGICAL "DON'TS" (AND "DO'S"), by C. Hamilton Whiteford, M.R.C.S., L.R.C.P.; 1923. London: Harrison and Sons, Limited; Crown 8vo., limp cover, pp. 46. Price: 3s. net.

Medical Appointments.

DR. A. F. JOLLEY (B.M.A.) and DR. F. L. UTBER (B.M.A.) have been appointed Government Medical Officers at Leeton and at Cooma, respectively, in New South Wales.

* * * *

THE Board of Health of New South Wales has authorized the undermentioned to act as Inspectors under the *Cattle Slaughtering and Diseased Animals and Meat Act, 1902*: DR. K. K. SPENCE (B.M.A.) and DR. A. TARLETON (B.M.A.) at Woodstock and at Baradine, respectively.

* * * *

DR. E. T. HILLIARD (B.M.A.) has been appointed Resident Medical Officer in the Department of Mental Hospitals of New South Wales.

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THE appointments have been confirmed of DR. ALICE W. MCCLOY and DR. DAISY H. RATCLIFF as Medical Officers in the Medical Branch of the Department of Education of New South Wales.

* * * *

DR. J. H. BLACKBURN (B.M.A.) has been appointed Public Vaccinator at Clunes, Victoria.

* * * *

DR. D. BROWNE has been appointed Acting Chief Resident Medical Officer, Sanatorium, Wooroloo, Western Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY: Resident Pathologist.

ROYAL HOSPITAL FOR WOMEN, SYDNEY: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B. M. A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

AUG. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
AUG. 30.—South Australian Branch, B.M.A.: Branch.
AUG. 31.—New South Wales Branch, B.M.A.: Branch.
SEP. 5.—Victorian Branch, B.M.A.: Branch.
SEP. 6—New South Wales Branch, B.M.A.: Nomination of two candidates to Federal Committee.
SEP. 7.—Queensland Branch, B.M.A.: Branch.
SEP. 11.—New South Wales Branch, B.M.A.: Ethics Committee.
SEP. 12.—Western Australian Branch, B.M.A.: Council.
SEP. 12.—Melbourne Pædiatric Society.
SEP. 14.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEP. 14.—Queensland Branch, B.M.A.: Council.
SEP. 14.—South Australian Branch, B.M.A.: Council.
SEP. 14.—Central Southern Medical Association, New South Wales.
SEP. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEP. 19.—Victorian Branch, B.M.A.: Council.
SEP. 19.—Western Australian Branch, B.M.A.: Branch.
SEP. 19.—South Sydney Medical Association, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

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